Brain Matters

UNDERGRADUATE NEUROSCIENCE | JOURNAL

VOLUME VIII

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The Impact of GLP-1 Receptor Agonists on the Brain's Addiction and Satiety Networks

Written by Noreen Adoni

Abstract

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs), with one of the most recognized being Ozempic, have increased in popularity for their abilities to help individuals with obesity or type 2 diabetes experience weight loss results. These medications mimic the GLP-1 hormone secreted by intestinal L-cells in response to the ingestion of a meal to slow gastric emptying, help regulate blood glucose levels, and promote feelings of fullness. A large aspect of their success in stimulating weight loss comes from their ability to bind to GLP-1 receptors in the hypothalamus, which stimulates satiety by activating neurons that produce satiety signals while inhibiting neurons that promote hunger, lowering overall food consumption and reducing appetite as a result. The impact that altering feeding networks in the brain can have on weight management illustrates the strong correlation between levels of food consumption and psychological aspects of hunger and fullness. While GLP-1 RAs are effective in providing weight loss results, they often come with adverse effects, such as nausea and headaches. Therefore, understanding the mechanisms behind GLP-1 RAs in the brain altering feeding behavior may help to develop future treatments that have a similar function in stimulating weight-loss but do not result in harmful side effects.

Increased Prevalence of GLP-1 RAS

Ozempic, one of the most well-known glucagon-like peptide-1 receptor agonist (GLP-1 RA) drugs on the market, has been referred to in the media as a "miracle drug" due to its ability to help individuals who have struggled with their weight for the majority of their lives finally achieve weight loss results. Obesity, defined as a BMI greater than or equal to 30, is a crucial issue in the United States. In 2017-2018, the age-adjusted prevalence of obesity in adults was 42.4%, increasing by 12% since 2000 (Hales et al., 2020). According to the National Institute of Diabetes and Digestive and Kidney Diseases, nearly one in three adults are overweight, and more than two in five adults are obese (NIDDK, n.d.). Obesity can have a serious impact on health, leading to heart disease, stroke, type two diabetes, musculoskeletal disorders, and certain cancers, which all contribute to premature death and substantial disability (World Health Organization, 2024). Therefore, obesity is a long-standing problem that severely endangers the health of a large portion of society. GLP-1 RAs, such as Ozempic, have been found to help individuals with obesity experience weight loss results, and a crucial reason for their success can be credited towards their ability to impact the appetite and satiety networks in the brain, altering feeding behavior.

In the past decade, many individuals have turned to GLP-1 RAs desperate for a solution to their struggle with weight. Use of these drugs has increased by 40-fold between 2017 and 2021, and six million Americans are now on either Ozempic or Mounjaro, which is another class of GLP-1 medication. From 2018 to 2023, 1,063,200 patients were prescribed a GLP-1 drug (Gratzl et. al, n.d.). An estimated nine million prescriptions were written in 2022, and roughly 2-3% of the United States population may now be taking one of these drugs (Logan, 2024). These statistics illuminate the increased prevalence of and dependency on GLP-1 RAs in recent years to combat weight issues.

Although GLP-1 RAs are commonly used for and understood as effective in stimulating weight-loss, there is a risk of experiencing side-effects when administered these medications. Several case reports have linked the use of these drugs with the occurrences of acute kidney injury, nausea, injection site reactions, headache, and nasopharyngitis (Filippatos et. al., 2014). Therefore, it is crucial to assess how GLP-1 RAs function in the brain to alter feeding behavior to eventually develop similar treatments that can suppress hunger and relay satiety signals to stimulate weight-loss without causing these harmful effects.

GLP-1 RA Mechanisms

GLP-1 RAs alleviate obesity by mimicking the action of glucagon-like peptide, a hormone secreted by the small intestine. Glucagon-like peptide triggers insulin, which is an essential hormone released from the pancreas that allows the body to use food for energy by lowering the amount of glucose in the blood (Cleveland Clinic, 2023). This extra insulin stimulated by a GLP-1 RA helps lower blood sugar levels, which is helpful for controlling type 2 diabetes and obesity. GLP-1 RAs also curb hunger by slowing the movement of food from the stomach into the small intestine, resulting in the body releasing less glucose from food into the bloodstream and allowing the individual to feel full faster and for longer. It is currently not completely understood why obese patients secrete less GLP-1 (Castro, 2022).

While these effects of GLP-1 RA drugs are crucial to their success in causing weight loss, the ability of GLP-1 RAs to influence the central nervous system's regulation of appetite and satiety is a major reason for their effectiveness. GLP-1 drugs exert their effects on glucose homeostasis and feeding behavior via indirect and direct pathways that the central nervous system mediates (Bloemendaal et. al., 2014). Hunger can be viewed as an addiction, as it is a learned behavior that eating is initially reinforcing by reversing an unpleasant bodily signal, such as changes in nutrient levels in the blood, changes in hunger hormones, and stomach contractions (Dagher, 2009). Altering and observing the brain's addiction and satiety networks can help an individual with a hunger or food addiction control their feeding behaviors. Therefore, a large reason for the effectiveness of GLP-1 drugs for weight loss is their ability to address the psychological factors that lead to obesity, impacting the brain's addiction and satiety networks in order to alter feeding behavior.

Regulation of Appetite and Satiety

Multiple parts of the hypothalamus, including the ventromedial nuclei, lateral hypothalamic area, and arcuate nucleus, work together to regulate appetite and satiety. Feelings of hunger and fullness involve complex interactions between hormones from the gastrointestinal tract to the hypothalamus. Ghrelin and leptin are two hormones that frequently signal to the hypothalamus to regulate sensations of hunger and satiety and to maintain energy homeostasis by balancing energy intake and expenditure. First, ghrelin, known as the hunger hormone, is produced by the gut and acts on the lateral hypothalamus. Ghrelin interacts with the growth hormone secretagogue receptor to promote feelings of hunger and food anticipation. Conversely, leptin, which is produced from adipose tissue, is the body's satiety signal and acts upon the arcuate nucleus, ventromedial nucleus, and lateral hypothalamus to promote stimulatory effects of satiety and inhibitory effects of hunger to coordinate the body's energy homeostasis. Together, ghrelin and leptin signals regulate sensations of hunger and satiety. Additional signals released from the gut,such as short-acting cholecystokinin and long-acting incretin, work to inhibit appetite in order to regulate energy homeostasis (Yeung and Tadi, 2023). In particular, GLP-1 drugs specifically mimic the long acting incretin-GLP signal to inhibit appetite. Glucagon-like peptide-1 (GLP-1) belongs to a family of hormones called incretins, which enhance the secretion of insulin. GLP-1 is synthesized and secreted by L-cells of the small intestine in response to food intake. It is also synthesized by a small population of neurons in the hunger center nucleus of the solitary tract (NTS) in the caudal brainstem, with the NTS projecting to areas in the hypothalamus and hindbrain, such as the arcuate nucleus, that express GLP-1 receptors (Barakat et al., 2024). When food is ingested, the vagus nerve relays satiety signals from GLP-1s that are secreted by L-cells to the GLP-1 receptors in the hypothalamus, with the purpose of controlling food intake. GLP-1 receptors in the hypothalamus stimulate fullness by activating neurons that produce satiety signals, such as pro-opiomelanocortin and cocaine and amphetamine-regulated transcript, while reducing food intake by inhibiting neurons that promote hunger, such as neuropeptide Y and agouti-related peptide (Baggio and Drucker, 2014).

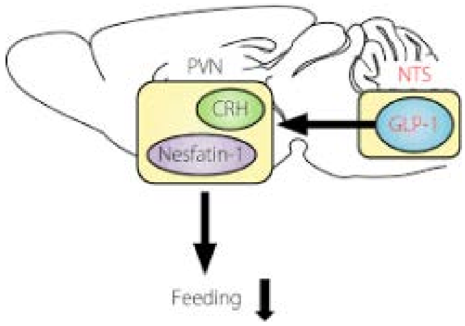


Figure 1. This model represents the interaction between GLP-1 sent from the NTS to GLP-1 receptors in the paraventricular nucleus within the hypothalamus, resulting in feeding suppression (Katsurada & Yada, 2016).

These results can also be observed when GLP-1 receptor agonists (GLP-1 RAs) are made to bind to GLP-1 receptors, mimicking the effects of GLP-1. GLP-1 RAs are administered subcutaneously, ensuring rapid absorption and peak concentration within hours. Post absorption, GLP-1 RAs exhibit a low volume of distribution and primarily remain in the bloodstream. These agents then selectively target GLP-1 receptors in various tissues involved in glucose regulation, with specific affinities for pancreatic cells and other metabolic control sites (Collins and Costello, 2024). In particular, by binding to GLP-1 receptors in the hypothalamus, GLP-1 RAs adapt the ability of GLP-1 to help control food intake and satiety.

Impact of GLP-1 RAS on Feeding Behavior Demonstrated

The effectiveness of GLP-1RAs on feeding behavior has been demonstrated in multiple studies. For example, a study performed by Friedrichsen and colleagues in 2021 demonstrated the influence of GLP-1 RAs, specifically semaglutide, on feeding habits and hunger levels. A group of seventy two adults with obesity were randomized with either taking a once-weekly semaglutide of 2.4 milligrams or a placebo for twenty weeks. Gastric emptying was assessed following a standardized breakfast, in addition to energy intake during lunch being examined. Researchers also assessed participants' appetite ratings and responses from a "control of eating" questionnaire, which prompted participants to note their eating behaviors and feelings around food during the study. Results demonstrated that participants who took semaglutide experienced reduced hunger and food consumption and increased fullness and satiety compared to the placebo group. Their responses to the "control of eating" questionnaire indicated better self-control regarding eating and fewer and weaker food cravings compared to the placebo group responses. Body weight was reduced by 9.9% with semaglutide use and 0.4% with the placebo (Friedrichsen et al., 2021). Therefore, Friedrichsen and colleagues concluded that a once-weekly administration of semaglutide suppressed appetite, improved self-control regarding eating, and reduced food cravings, illustrating the effect of GLP-1 RAs on feeding behavior.

Since GLP-1 RAs are effective in altering feeding behavior and decreasing appetite, individuals who stop taking GLP-1 medications typically re-gain a majority of the weight they lost while taking the drug. This result is due to the secretion of GLP-1 and its binding to GLP-1 receptors both returning to pre-treatment levels once GLP-1 RAs are no longer administered, resulting in greater feelings of hunger and less satiety (Wilding et. al., 2022). This re-gaining of weight when administration of a GLP-1 RA is terminated demonstrates the ability of these medications to significantly alter feeding behavior and the regulation of satiety and hunger.

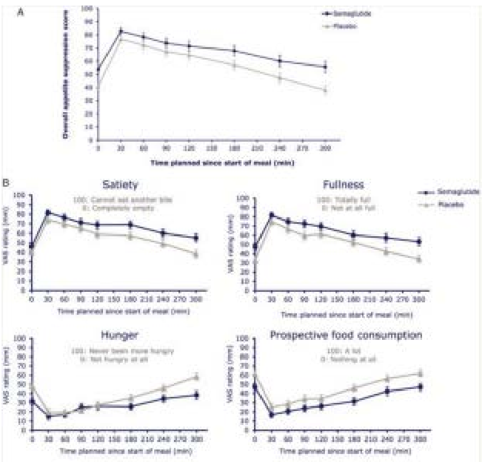


Figure 2. Results from the study performed by Blundell and colleagues, which randomized thirty subjects to a once-weekly dosage of semaglutide or placebo. Results show that overall appetite is lower and that satiety is higher in subjects who took the semaglutide (Blundell et. al., 2017).

Conclusion

The abilities of GLP-1 receptor agonists to mimic GLP-1 in individuals with less endogenous GLP-1 secretion, bind to GLP-1 receptors, and relay satiety signals to the hypothalamus to control feeding behavior, are what make these medications highly sought after by individuals seeking weight loss results. The impact of GLP-1 medications on the brain to alter feeding behaviors demonstrates that levels of food consumption is highly contingent on the ability of satiety signals to reach the brain. Therefore, GLP-1 RAs do not just work by slowing down gastric emptying to stimulate a feeling of fullness, but also directly impact the appetite centers in the brain by binding to GLP-1 receptors and relaying satiety signals. The direct impact of GLP-1 RAs on the brain by altering feeding behavior may be overlooked, but is demonstrated through the weight gain effects following termination of administration of the GLP-1RAs and subsequent increased appetite. Given that GLP-1 levels are lower in obese patients, future endeavors may seek to investigate the reasons for and mechanisms behind reduced secretion of GLP-1 in obese individuals. If the mechanisms behind the reduced GLP-1 secretion in obese patients are more fully understood, then treatments that target appetite centers and the reception of satiety signals in the brain may be developed that reduce the adverse withdrawal and weight-gain effects experienced when coming off GLP-1 RA medications. Trials on medications similar to GLP-1 RAs or that similarly target the appetite centers of the brain may also be conducted. Regardless, future research regarding medications that affect the appetite centers of the brain such as GLP-1RAs are limited to the difficulties of studying the in-vivo brain. By first fully understanding the complex mechanisms regarding satiety and feeding behavior in live individuals without invasive procedures, safer and more effective medications may be developed that permanently improve the quality of life for all.

References

1. Baggio, L. L., & Drucker, D.J. (2014). "Glucagon-like peptide-1 receptors in the brain: Controlling food intake and body weight." Journal of Clinical Investigation, 124(10), 4223-4226. https://doi.org/10.1172/jci78371

2. Barakat GM, Ramadan W, Assi G, Khoury NBE. Satiety: "A gut-brain-relationship." J Physiol Sci. 2024 Feb 17;74(1):11. doi: 10.1186/s12576-024-00904-9. PMID: 38368346; PMCID: PMC10874559.

3. Blundell, J., Finlayson, G., Axelsen, M., Flint, A., Gibbons, C., Kvist, T., & Hjerpsted, J. B. (2017b). "Effects of once-weekly semaglutide on appetite, energy intake, control of eating, food preference and body weight in subjects with obesity." Diabetes, Obesity and Metabolism, 19(9), 1242–1251. https://doi.org/10.1111/dom.12932

4. Cleveland Clinic Medical. (2024, October 11). "GLP-1 agonists." Cleveland Clinic. https://my.clevelandclinic.org/health/treatments/13901-glp-1-agonists

5. Collins, L. (2024, February 29). "Glucagon-like peptide-1 receptor agonists." National Library of Medicine. https://www.ncbi.nlm.nih.gov/books/NBK551568/

6. Dagher, A. "The neurobiology of appetite: hunger as addiction." Int J Obes 33 (Suppl 2), S30–S33 (2009). https://doi.org/10.1038/ijo.2009.69

7. Fillippatos, T. D., Panagiotopoulou, T. V., & Elisaf, M. S. (2015). "Adverse Effects of GLP-1 Receptor Agonists." https://doi.org/10.1900/RDS.2014.11.202.

8. Friedrichsen, M., Breitschaft, A., Tadayon, S., Wizert, A., & Skovgaard, D. (2021). "The effect of semaglutide 2.4 mg once weekly on energy intake, appetite, control of eating, and gastric emptying in adults with obesity." Diabetes, Obesity and Metabolism, 23(3), 754–762. https://doi.org/10.1111/dom.14280

9. Gratzl, S., Rodriguez, P. J., Cartwright, B. M., Baker, C., & Stucky, N. L. (2024a). "Monitoring report: GLP-1 RA prescribing trends - September 2024 data." https://doi.org/10.1101/2024.01.18.24301500

10. Hales, C. M., Carroll, M. D., Fryar, C. D., & Ogden, C. L. (2020, February 27). "Prevalence of Obesity and Severe Obesity Among Adults: United States, 2017-2018." February 2020. Centers for Disease Control and Prevention. https://www.cdc.gov/nchs/products/databriefs/db360.htm

11. Katsurada, K., & Yada, T. (2016). "Neural effects of gut- and brain-derived glucagon-like peptide-1 and its receptor agonist." *Journal of Diabetes Investigation, 7(*S1), 64-69. https://doi.org/10.1111/jdi.12464

12. National Institute of Diabetes and Digestive and Kidney Diseases. (n.d.-a). "Overweight & Obesity Statistics." Niddk. https://www.niddk.nih.gov/health-information/health-statistics/overweight-obesity

13. Pierce, L. (2024, June 27). "On the increase in use of GLP-1s." https://medicine.iu.edu/blogs/bioethics/on-the-increase-in-use-of-glp-1s

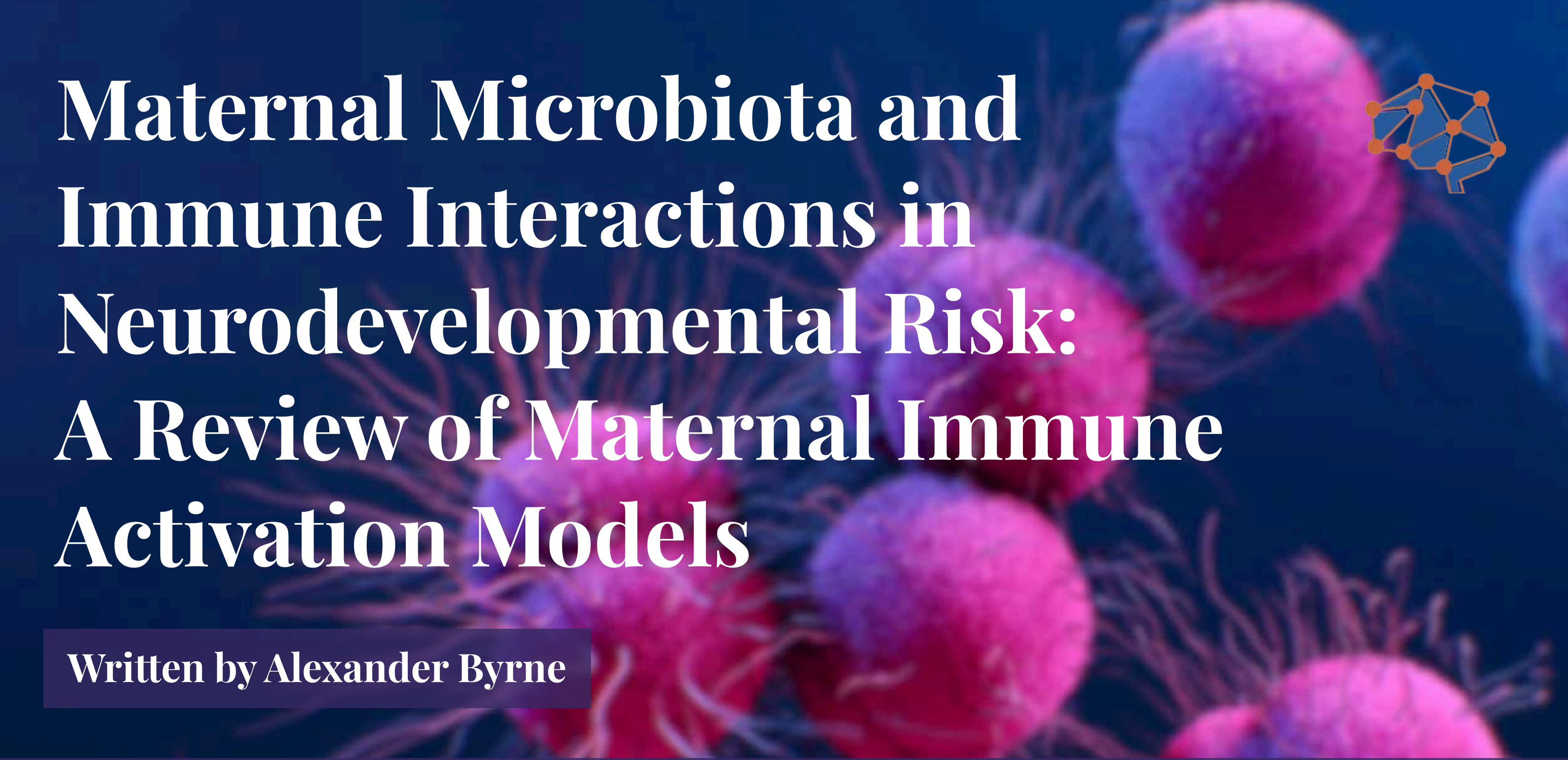
14. Van Bloemendaal, L., ten Kulve, J. S., la Fleur, S. E., Ijzerman, R. G., & Diamant, M. (2013b). "Effects of glucagon-like peptide 1 on appetite and body weight: Focus on the CNS." Journal of Endocrinology, 221(1). https://doi.org/10.1530/joe-13-0414

15. Wilding, J. P., Batterham, R. L., Davies, M., Van Gaal, L. F., Kandler, K., Konakli, K., Lingvay, I., McGowan, B. M., Oral, T. K., Rosenstock, J., Wadden, T. A., Wharton, S., Yokote, K., & Kushner, R. F. (2022). "Weight regain and cardiometabolic effects after withdrawal of semaglutide: The step 1 trial extension." Diabetes, Obesity and Metabolism, 24(8), 1553–1564. https://doi.org/10.1111/dom.14725

16. Yeung, A. Y. (2023, January 3). "Physiology, Obesity, Neurohormonal Appetite and Satiety Control." National Library of Medicine. https://www.ncbi.nlm.nih.gov/books/NBK555906/

About the Author

Noreen is a freshman at the University of Illinois majoring in Neuroscience. She joined Brain Matters to investigate how the brain impacts the ways in which we interact with the world around us and stay updated on current research in the field. Noreen is interested in studying neurological diseases, hoping to further analyze and treat them as a physician in the future.



Maternal Microbiota and Immune Interactions in Neurodevelopmental Risk: A Review of Maternal Immune Activation Models

Written by Alexander Byrne

Abstract

Maternal Immune Activation (MIA) is a model used to study the impact of maternal infection and maternal inflammatory cues on fetal brain development and vulnerability to neurodevelopmental disorders (NDDs). Studies using the MIA model have confirmed the role of immune mediators, such as interleukin-17A (IL-17A), in disrupting the cortex's structural integrity and behavior. The maternal microbiota has also been recognized as a key modulator of these immune-fetal brain interactions. Microbes that inhabit the gut control TH17 cell differentiation—long-term players in MIA—and presence or absence can determine whether offspring become NDD-like following maternal inflammation. This association suggests the capacity of the microbiota to modulate maternal immune reactivity and thereby determine fetal neurodevelopmental processes. Together, these findings position MIA not as a singular immune event but as a systems-level interaction among microbes, cytokines, placental signaling, and fetal neuroimmune development.

Introduction

Neurodevelopmental disorders (NDDs) are some of the most notable and commonly discussed disorders in neuroscience. These disorders are dependent on prenatal environmental changes and are often studied in rodent models, though establishing one-to-one comparisons between rodents and humans remains difficult. While rodent models exhibit NDD-like symptoms, their mapping to human conditions is tentative. Significant stages of development are the prenatal and early postnatal periods, which are particularly sensitive to severe viral infections and the resulting immune response (Chalen, Caetano-Silva, et al., 2024; Otero and Antonson, 2022). This response can contribute to the development of NDD-like symptoms. The model used to study this interaction is known as Maternal Immune Activation (MIA), which occurs when a pregnant mother's immune system is triggered by infection, leading to changes in fetal brain development (Otero and Antonson, 2022; Chalen, Caetano-Silva, et al., 2024). The immune system plays a vital role in protecting the body from infection and disease, yet it can also have adverse effects. Research indicates that certain cell populations involved in immune responses can produce unexpected neurological side effects on fetal offspring. Beyond infection and immune response, other external factors, such as the maternal microbiome, have also been linked to NDDs. This review aims to understand the relationship between the maternal microbiome and the phenomenon of MIA.

Modeling MIA with Poly I:C

The study by Choi and collaborators demonstrates how gut microbes regulate maternal immune responses and influence fetal brain development (Choi et al., 2016). The molecule used to study this is Poly I: C, which was used to activate the immune system artificially, distinct from a live virus. This triggers the cell populations of the immune response known as T Helper 17 cells (TH17 cells), which are shown to be responsible for ASD-like symptoms in rodent offspring. Looking deeper into this, the effector cytokine interleukin-17a (IL-17a) is directly related to the microbiome and is necessary and sufficient to cause abnormal behaviors and brain development in the rodent model (Choi et al., 2016). The maternal interleukin-17a pathway in mice promotes autism-like phenotypes in offspring. Normally, neurons in the cerebral cortex organize into distinct layers, but MIA leads to patches of disorganized neurons similar to those seen in ASD postmortem brains (Choi et al., 2016).

While it has been shown that MIA caused by infection can lead to the development of abnormal behaviors and brain development in rodent models' offspring, it remains to be seen the degree to which other factors, such as the microbiome, play as significant a role. During TH17 cell differentiation, the maternal microbiome and bacteria play an essential role. Segmented filamentous bacteria (SFB) are strongly associated with TH17 production and activity, and mice treated with vancomycin lack this but recover it when in contact with non-treated mice/waste (Kim et al., 2017). When treated with broad-spectrum antibiotics or minimally possessing TH17 cells in the small intestine, cortical patches consistent with ASD-like morphology in fetal offspring decreased. Therefore, maternal gut bacteria are indispensable to promote neurodevelopmental abnormalities in mouse offspring (Kim et al., 2017). Two genetically similar strains of mice, Taconic (Tac) and Jackson (Jax), differ in their TH17 cell populations due to the presence or absence of SFB. When subjected to MIA, Tac offspring display NDD-like behaviors, whereas Jax offspring do not. However, when Jax mothers are colonized with SFB, their offspring develop ASD-like behaviors, demonstrating that maternal gut bacteria influence neurodevelopment. Pregnant Jax mice colonized with human gut bacteria that promote TH17 cells also develop ASD-like offspring after MIA, reinforcing the idea that human microbiota may contribute to neurodevelopmental abnormalities.

Building on the IL-17A mechanism, Shin Yim and collaborators (Shin Yim et al., 2017) demonstrated that the neurodevelopmental outcomes of maternal immune activation (MIA) are not permanent and can indeed be reversed postnatally (Shin Yim et al., 2017). Utilizing a poly I:C model to mimic viral infection in pregnancy, offspring exhibited typical neurodevelopmental disorder (NDD)-like behaviors, such as decreased social interaction, augmented repetitive behaviors, and hyper ultrasonic vocalizations. These behaviors had already been linked to cortical disorganization in the form of loss of layer specific markers like SATB2 and TBR1 (Shin Yim et al., 2017). Notably, the study localized these defects to discrete cortical patches—primarily in the dysgranular zone of the primary somatosensory cortex (S1DZ)—and linked their presence and size with the severity of behavior. In Figure 1f, the S1 of MIA offspring had elevated c-Fos expression, indicative of elevated baseline neuronal activity, again implicating hyperexcitable cortical circuits as an etiology for the phenotypes. In a tour de force, the investigators then proceeded to employ postnatal intervention by optogenetically silencing this overactive cortical region, rescuing both behavior and cortical anatomy. Importantly, the behavioral rescue was most effective when performed during the early postnatal time points, emphasizing an early window of enhanced neuroplasticity. These findings contend that while MIA imposes structural and functional changes on fetal brain development, these changes are not permanent but rather are therapeutically accessible by virtue of neural activity manipulation within key cortical nodes like the S1DZ (Shin Yim et al., 2017).

Modeling MIA with Real Pathogens: The Use of Live Viruses in Research

The review by Otero and Antonson (Otero and Antonson, 2022) highlights maternal immune activation (MIA) as a multifaceted model for understanding how prenatal infections shape fetal neurodevelopment (Otero and Antonson, 2022). Central to their argument is the distinction between commonly used pathogen mimetics like poly I: C and live virus models such as influenza A virus (IAV) (Otero and Antonson, 2022). Whereas poly I:C triggers a brief, acute innate immune response, IAV triggers a long-term, multi-stage immune cascade along both innate and adaptive pathways of immunity. The paper notes that although both models activate the TH17/IL-17A pathway—a pathway previously demonstrated to interfere with cortical morphology and stimulate fetal microglia—IAV more accurately reflects in vivo infections. Importantly, they argue that poly I: C may oversimplify MIA by bypassing key interactions between maternal microbes, cytokines, and fetal immune cells. The review also underscores additional mechanisms, such as placental lack of oxygen and microglial priming, which could potentially operate independently or alongside IL-17A signaling.

Antonson and collaborators in their 2021 study provide critical insight into the threshold model of fetal vulnerability by studying the effects of moderately pathogenic IAV infection during pregnancy (Antonson, Kenney, et al., 2021). Pathogenicity is a major player in the MIA model, and the degree of infection is a crucial factor to consider. According to this study, moderate infection might have muted or even negligible effects on cortical formation and fetal brain inflammation, though there is a noted impact on placental health (Antonson, Kenney, et al., 2021). This observation underscores the important role of the placenta as a protective barrier against maternal inflammation (Antonson, Kenney, et al., 2021). Typical immune responses with moderate doses include systemic cytokine elevations, such as IL-6, albeit to a lesser extent than those seen with high-dose infections. Additionally, while cytokines in the placenta may lead to structural integrity breakdown, the absence of a fetal brain response suggests that the placenta acts as the first line of defense, a notion further supported by pathway analyses showing upregulation of inflammatory and hypoxia-related genes (Antonson, Kenney, et al.).

In their 2024 study, Otero and collaborators explored how IAV infection during pregnancy affects fetal brain development in a dose- and time-dependent manner (Otero et al., 2024). Severity has been demonstrated to be a significant factor when considering the infection type for the MIA model. Although the model often focuses on IL-17A and TH17 pathways, activation by IAV does not elevate these components as one might suppose; instead, fetal microglia and border-associated macrophages emerge as the primary responders, increasing in both number and phagocytic behavior. While cytokines such as IL-6 and IFN-γ consistently rise with the level of infection—a constant feature of MIA— maternal IL-17A and TH17 cell numbers remain relatively constant. Consequently, high dose IAV infection results in a thinning of the cortical plate and disorganization of both deep and upper cortical layers, evidenced by altered distributions of TBR1+ and SATB2+ neurons (Otero et al., 2024). This disruption is absent in moderate IAV infection, further reinforcing the concept of a severity threshold. High-dose infection also alters gene regulation for neuronal development, inflammation, and microglial function, indicating that severe maternal inflammation disrupts normal cortical development.

The Microbiome and MIA

Other factors such as stressors, metabolites and others contribute to the complexity of the MIA model and the neurological realities within. Antonson and collaborators in their 2020 study investigated how prenatal stress alters both maternal immune function and gut microbiota composition (Chen, Antonson, et al., 2020). The study found that stress during gestation led to unique immune signatures in pregnant dams, including elevated cytokines such as IL-6 and CCL2, alongside shifts in microbial diversity. These changes were linked to disrupted microbial metabolic pathways, which influence neuroactive compound production. The study underscores the importance of the microbiome as a mediator between psychological stress and systemic immune activation during pregnancy. The altered microbial and immune landscape suggests a possible route by which prenatal stress exerts long-term effects on offspring brain development.

Chen and collaborators in their 2020 study similarly examined prenatal stress's effects on initiating disturbances in the immune system and neurochemistry with long term behavioral impacts in offspring (Chen, Antonson, et al., 2020). From their findings, prenatal stress generated intrauterine inflammation characterized by increased CCL2 expression that, in turn, affected serotonin signaling pathways responsible for neurodevelopment. Behavioral tests revealed that such offspring exposed to this inflammatory microenvironment had enduring impairments of anxiety-like behavior and social interaction.

“The altered microbial and immune landscape suggests a possible route by which prenatal stress exerts long-term effects on offspring brain development.”

Notably, these behaviors were dependent on both microbial colonization and CCL2 signaling, indicating that stress-induced behavioral phenotypes are regulated by microbiota-immune crosstalk. Galley and collaborators in their 2021 study studied the ways in which prenatal stress regulates tryptophan metabolism, a critical connection between the gut microbiome and neurodevelopment (J. Galley et al.,2021). The study demonstrated that stress during pregnancy disrupted both microbial and host tryptophan metabolic pathways, including those leading to the synthesis of serotonin and kynurenine. These were accompanied by alterations in fetal brain and placental tryptophan transporter expression, showing that maternal stress can impair neurotransmitter supply at critical developmental windows. The data suggests a mechanism where the maternal microbiome influences neurodevelopment via metabolite signaling, adding a biochemical dimension to the immune-mediated MIA framework.

Galley and collaborators in their 2023 study examined how other stressors impact developing offspring gut microbiomes (Galley et al., 2023). Using human cohorts and mouse models, the study revealed that high maternal psychological distress was associated with reduced bifidobacteria abundance and decreased microbial richness in offspring. Extending this work, (Galley et al., 2024) studied the effectiveness of probiotic intervention by modulating *Bifidobacterium dentium* administration during pregnancy (Galley et al., 2024). The study demonstrated that prenatal exposure to this specific strain had long-term intergenerational effects, including altered immune profiles, metabolic signaling, and improved social behaviors in offspring. Offspring of *B. dentium*-treated dams exhibited reduced pro-inflammatory cytokine expression and more balanced microbiota composition. These findings are significant in that they show intentional manipulation of maternal microbiota can have trans-generational protective effects.

Discussion and future directions

Taken together, these reports support a multi-aspect interaction among maternal stress, microbial ecology, immune signaling, and neurodevelopment of the offspring. In their early phase, MIA experiments focused mostly on cytokines such as IL-6 and IL-17A, but emerging evidence hints toward the universal involvement of maternal microbiota in eliciting immunity and programming fetal fates. Pioneering experiments by (Choi et al., 2016) and (Kim et al., 2017) showed that gut microbiota in mothers—here specifically those triggering TH17 differentiation—are required to trigger pathogenic cascades that lead to cortical defects and autism-like behaviors in offspring (Choi et al, 2016.; Kim et al., 2017). However, recent work by (Otero et al., 2024) and (Antonson, Kenney, et al., 2021) has opened the paradigm using live influenza A virus (IAV) models, revealing immune dynamics that encompass placental hypoxia, microglial priming, and a threshold of severity for fetal brain effects—none of which are fully captured in synthetic mimetic models like poly I:C (Antonson, Kenney, et al., 2021; Otero et al., 2024). Other recent research has tried to explore the role of IL17A in the absence of microbes (Chalen, Wang, Jung, et al. 2022; Chalen, Wang, Florianowicz, et al., 2023).

In parallel, studies by Chen, Galley, and Antonson have extended this framework beyond infection, showing that non-infectious stressors such as psychological distress during pregnancy also disrupt maternal microbial communities and immune tone (Antonson, Evans, et al, 2020.; Galley et al, 2024.; Chen, Galley, et al., 2021). These disruptions—marked by altered tryptophan metabolism, inflammatory chemokine expression, and reduced bifidobacterial abundance—contribute to long-term changes in neurodevelopmental signaling pathways and behavior. Notably, (Galley et al., 2024) demonstrate that probiotic interventions using B. dentium during gestation can reverse or mitigate these effects, leading to improved social behavior and reduced inflammation in offspring, even across generations (Galley et al., 2024). This body of work supports a more integrative and systems-level approach to understanding MIA. Future studies should holistically account for the interactions among maternal microbial ecology, immune response, infection severity, psychological stress, and critical windows of neurodevelopment. COVID-19 in pregnant women acts as an example of MIA and has already been shown to cause the development of NDD-like symptoms (Duan et al., 2024). While the mechanism hasn't been figured out, the expansive scientific literature and research on influenza will likely inform future research pathways regarding Covid-MIA interactions. Future research will look at interactions between the microbiome and influenza infection in the mouse model and how both relate to the development of NDD.

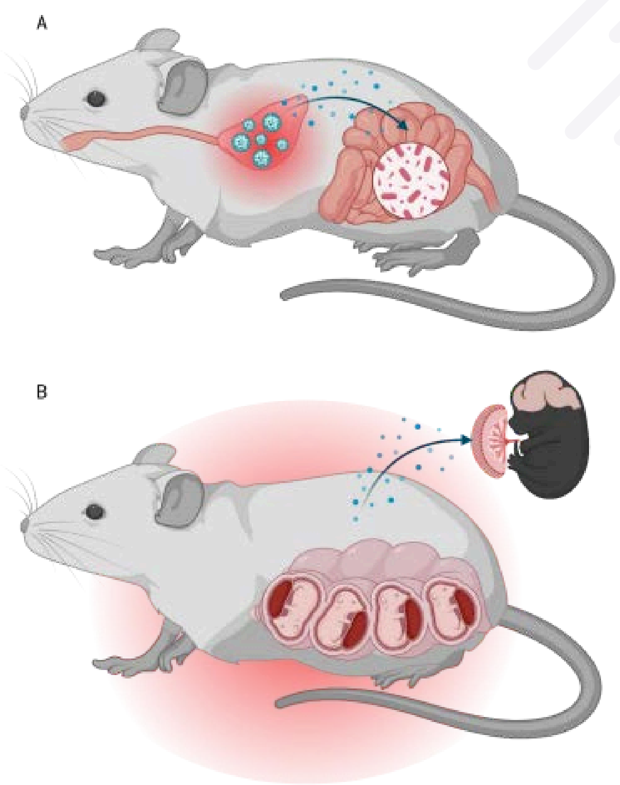


Figure 1. (A) Maternal respiratory infection can cause systemic inflammation and disrupt gut microbiota. (B) Maternal systemic inflammation can be detrimental to fetal neurodevelopment. (Source: BioRender)

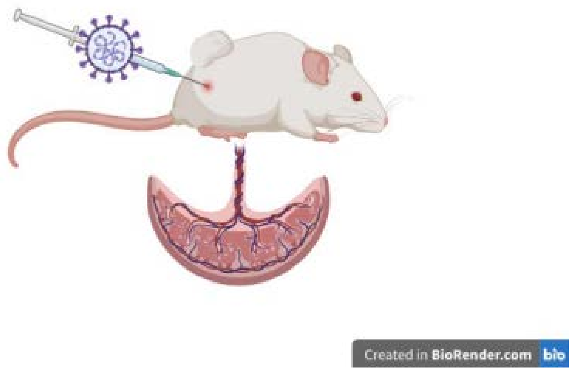


Figure 2. Infected Mouse and Placental Changes: (a) Pregnant Mice are innoculated with influenza on Gestational Day (GD) 9.5 and sacked and placentae extracted at GD 16.5. Placental integrity and morphology are then measured. (Source: BioRender)

Acknowledgments: I would like to acknowledge the members of the Antonson Developmental Neuroimmunology Laboratory: Izan Chalen, Dr Adrienne Antonson, Rafael Gonzalez-Ricon, Ashley Otero, Arnav Kaushik, Fernando Rigal, Hao-Chun Fan, and my family: Christy Beighe-Janke, Vincent Byrne, Harmony Byrne, Miracle Byrne, Cassidy Byrne, and Margaret Byrne.

References

1. Antonson, Adrienne M., Adam D. Kenney, et al. "Moderately Pathogenic Maternal Influenza A Virus Infection Disrupts Placental Integrity but Spares the Fetal Brain." *Brain, Behavior, and Immunity*, vol. 96, Aug. 2021, pp. 28–39, https://doi.org/10.1016/J.BBI.2021.05.004.

2. Antonson, Adrienne M., Morgan V. Evans, et al. "Unique Maternal Immune and Functional Microbial Profiles during Prenatal Stress." *Scientific Reports 2020 10:1*, vol. 10, no. 1, Nov. 2020, pp. 1–15, https://doi.org/10.1038/s41598-020-77265-x.

3. Chalen, Izan, Maria Elisa Caetano-Silva, et al. "Alterações Psiconeurológicas Do Desenvolvimento Relacionadas a Infecções Virais [Psychoneurological Alterations of Development Related to Viral Infections]." *Neuroinflamação: Da Biologia à Terapia [Neuroinflammation: From Biology to Therapy]*, edited by W. Savino et al., 2024.

4. Chalen, Izan, Selena Wang, Virginia Florianowicz, et al. "Measuring Fetal Cortical Architecture in Response to Maternal Immune Activation by Live Influenza Virus." *Brain, Behavior, and Immunity*, vol. 114, Nov. 2023, p. 31, https://doi.org/10.1016/J.BBI.2024.01.098.

5. Chalen, Izan, Selena Wang, Joseph Jung, et al. "Measuring Fetal Neuroarchitecture in Germ-Free Mice in Response to Maternal IL-17a Administration." *Brain, Behavior, and Immunity*, vol. 106, Nov. 2022, p. 20, https://doi.org/10.1016/J.BBI.2022.07.073.

6. Chen, Helen J., Jeffrey D. Galley, et al. "Fetal CCL2 Signaling Mediates Offspring Social Behavior and Recapitulates Effects of Prenatal Stress." *Brain, Behavior, and Immunity*, vol. 115, Jan. 2024, pp. 308–18, https://doi.org/10.1016/J.BBI.2023.10.032.

7. Chen, Helen J., Adrienne M. Antonson, et al. "Prenatal Stress Causes Intrauterine Inflammation and Serotonergic Dysfunction, and Long-Term Behavioral Deficits through Microbe- and CCL2-Dependent Mechanisms." *Translational Psychiatry 2020 10:1*, vol. 10, no. 1, June 2020, pp. 1–12, https://doi.org/10.1038/s41398-020-00876-5.

8. Choi, Gloria B., et al. "The Maternal Interleukin-17a Pathway in Mice Promotes Autism-like Phenotypes in Offspring." *Science*, vol. 351, no. 6276, Feb. 2016, pp. 933–39, https://doi.org/10.1126/SCIENCE.AAD0314/SUPPL_FILE/PAPV2.PDF.

9. Duan, Lian, et al. "Maternal COVID-19 Infection Associated with Offspring Neurodevelopmental Disorders." *Molecular Psychiatry*, vol. 30, no. 5, May 2024, pp. 2108–18, https://doi.org/10.1038/S41380-024-02822-Z;TECHMETA=38,39,47;SUBJMETA=378,476,631,692,699;KWRD=NEUROSCIENCE,PS YCHIATRIC+DISORDERS.

10. Galley, Jeffrey, et al. "Prenatal Stress in Mice Leads to Dysregulation in Maternal Microbial Tryptophan Metabolism and Downstream Signaling in Mom and Fetus." *Biological Psychiatry*, vol. 89, no. 9, May 2021, p. S286, https://doi.org/10.1016/J.BIOPSYCH.2021.02.712.

11. Galley, Jeffrey D, et al. "Gestational Administration of Bifidobacterium Dentium Results in Intergenerational Modulation of Inflammatory, Metabolic, and Social Behavior." *Brain, Behavior, and Immunity*, vol. 122, Nov. 2024, pp. 44–57, https://doi.org/10.1016/j.bbi.2024.08.006.

12. Galley, Jeffrey D., et al. "Maternal Anxiety, Depression and Stress Affects Offspring Gut Microbiome Diversity and Bifidobacterial Abundances." *Brain, Behavior, and Immunity*, vol. 107, Jan. 2023, pp. 253–64, https://doi.org/10.1016/j.bbi.2022.10.005.

13. Kim, Sangdoo, et al. "Maternal Gut Bacteria Promote Neurodevelopmental Abnormalities in Mouse Offspring." *Nature*, vol. 549, no. 7673, Sept. 2017, pp. 528–32, https://doi.org/10.1038/nature23910.

14. Otero, Ashley M., et al. "Influenza A Virus during Pregnancy Disrupts Maternal Intestinal Immunity and Fetal Cortical Development in a Dose- and Time-Dependent Manner." *Molecular Psychiatry*, July 2024, https://doi.org/10.1038/s41380-024-02648-9.

15. Otero, Ashley M., and Adrienne M. Antonson. "At the Crux of Maternal Immune Activation: Viruses, Microglia, Microbes, and IL-17A." *Immunological Reviews*, vol. 311, no. 1, Oct. 2022, pp. 205–23, https://doi.org/10.1111/IMR.13125.

16. Shin Yim, Yeong, et al. *Reversing Behavioural Abnormalities in Mice Exposed to Maternal Inflammation Characterization of Cortical Patches*. 2017, https://doi.org/10.1038/nature23909.

About the Author

Alexander Byrne, a native of the South Side of Chicago, has long fostered his fascination with science and narrative. Now a student pursuing a degree in Neuroscience, Alexander has focused his scholarly work on the overlap of maternal immune activation, neurodevelopmental disorders, and gut-brain microbiota interactions with the aim of determining how early immune signals influence the development of the brain and long-term behavioral outcomes.In addition to this study, he is deeply engaged in molecular neuroscience investigations into the structural processes of prion protein misfolding and aggregation. After finishing his undergraduate studies, Alexander plans to undertake a Ph.D. in neuroscience. His desire is to be involved in translational research that converts molecular biology into clinical knowledge.

Impact of Meditation on Brain Function

Written by Anika Chandola

Abstract

Meditation can be defined as a collection of mental training techniques aimed at regulating cognition, emotions, and the self. Rooted in spirituality, these practices take many forms in a variety of religions for some, or music and connection with nature for others. Western medical communities have begun to take a deeper look at this ancient phenomenon and have found it has several benefits, including the improvement of neuroplasticity, mental health, multifactorial diseases, and even age-related neurodegeneration. Specifically, Magnetic Resonance Imaging (MRI) has shown positive physical changes and introduced the idea of the Brain Theory of Meditation (BTM). This theory suggests that, through meditation, individuals can surpass previously assumed limitations of the human brain, increasing a multitude of cognitive functions. This change in physiology introduces a new field of discovery that calls for a reexamination of the inner workings of spirituality and health and what the potential for the human mind truly is.

Introduction

Meditation has become a modern focus in research after discovering the many benefits of this ancient practice, especially associated with the brain. Through the use of neuroimaging techniques, specifically EEG, researcher Julio Rodriguez-Larios has found that repetitive meditation practice can lead to increased activation of productive brain sites, strong neural pathways, and reconstruction of the brain over time (Rodriguez-Larios et al., 2021).

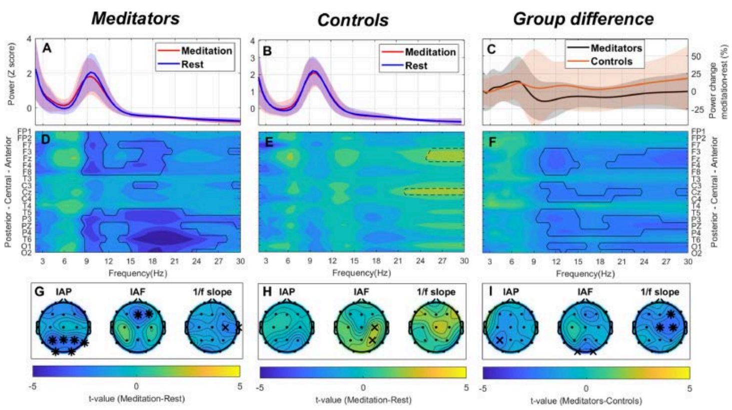


Figure 1. EEG spectral modulations associated with meditation in meditators and control groups (Rodriguez-Larios et al., 2021)

Through MRI experiments, more evidence suggests that over time meditation results in the activation of emotional and cognitive centers of the brain, specifically the prefrontal cortex, amygdala, and hippocampus, potentially lowering the risk for age-related neurodegenerative diseases and increasing cognition in young and middle-aged individuals (Davidson & McEwen, 2012) & (Newberg, 2001). Such drastic changes from a mental exercise suggest that thoughts affect our biology as seen in a slow down of telomerase shortening which increases the longevity of cells by preserving chromosomes throughout the aging process and implying an antiaging effect of meditation. (Epel, E. S., et al. 2009). Gray matter– which plays a critical role in processing information and regulating thoughts, memories, emotions, sensory input, and muscle movements–consists of high concentrations of neuronal bodies, axon terminals, and dendrites. More recent evidence shows that activities like meditation can boost the production of gray matter cells, challenging previous assumptions that brain cells stop developing after childhood. Studies have found that the brain's neuroplasticity continues into adulthood, meaning that the brain can grow and adapt, particularly through practices that stimulate cognitive and sensory engagement, such as mindfulness and other forms of meditation (Lazar et al., 2005; Hölzel et al., 2011). These findings highlight the brain's capacity for ongoing development and regeneration, even in later stages of life.

New studies indicate that meditation may also promote the growth of new neurons in areas of the brain associated with memory, emotion, and stress regulation, suggesting substantial advantages for both mental and physical health (Chaix, R., et al. 2017). Although the field is in its infancy, these discoveries offer promising opportunities for employing meditation not only as a means for mental clarity and relaxation but also as a technique that could enhance brain health at a cellular level. As researchers continue to explore these effects, meditation remains a promising and accessible practice with potential benefits that may surpass our existing understanding. In this paper, we will analyze the physiological effects of meditation on the brain, its long-term effects throughout the aging process, and how assorted meditations manifest in different effects in the brain.

Physiological Effect

In regards to studies conducted determining hot spots during meditation, neuroimaging studies have implied a few candidate regions that are most activated in continuous practice. An 8-week study using structural magnetic resonance imaging (MRI) on long-term mindfulness meditators revealed physical changes in brain structure (Hölzel et al. 2011). Notable findings included increased gray matter density and greater cortical thickness in brain regions associated with cognition, particularly in the posterior areas of the dorsal attention network (DAN). Other commonly activated regions include the precuneus, a region dedicated to Visuospatial processing, episodic memory retrieval, self-relevant processing, consciousness, and prefrontal regions of the default mode network (DMN), a notoriously large brain network most commonly active when the brain is at rest or not actively focused on the outside world. More longitudinal studies (Yang 2019), argue that the effects are more prevalent in longer-practicing meditators (people who meditate) and find more drastic physiological effects, in addition to a reduction in perceived stress and gray matter pathway strength, alluding to a correlation between improvement in mental emotional health and neuroplastic efficiency.

Meditation starts a chain of intricate neurobiological mechanisms that spread its advantages from the mind to the body. Research, exemplified by a study conducted by Hölzel et al. (2011), suggests that meditation can alter the structure and function of brain regions associated with stress, pain, and emotional control. Specifically, MRI scans revealed that engaging in mindfulness meditation can increase the thickness of the prefrontal cortex, a brain region involved in emotional control and decision-making. Improved function in the prefrontal cortex could potentially help the brain handle stress more effectively, leading to lower activation of the sympathetic nervous system, resulting in decreased blood pressure and reduced inflammation in the body.

During pain management, meditation influences the somatosensory cortex and insula, which are important in processing pain. Zeidan et al. (2011) showed that meditation decreased pain perception by influencing these brain regions, leading to less activity in the somatosensory cortex (related to feeling physical pain) and more activity in the anterior insula (connected to thinking about pain). This change assists the brain in reinterpreting pain signals, resulting in decreased pain perception and modifying chronic pain responses by reducing the brain's usual response to pain stimuli.

Moreover, meditation influences the brain's limbic system, particularly the amygdala, to help regulate the body's response to stress. Research conducted by Taren et al. (2013) discovered that participating in mindfulness training resulted in a decrease in the volume of the amygdala and a decrease in its activity when faced with stress. This change assists in controlling the release of cortisol from the hypothalamic-pituitary-adrenal (HPA) axis, which plays a crucial role in the body's response to stress. Decreased cortisol levels aid in cardiovascular health, enhance immune function, and decrease inflammation (Thau et al, 2023), demonstrating how meditation's impact on the brain's structure and function results in tangible physical health advantages in various bodily systems.

Meditation and Aging

Meditation’s most notable benefits are its prevention and improvement of Alzheimer’s, dementia, and other neurodegenerative diseases. Increasing life expectancy across the globe leads to increased cases of these neurocognitive diseases. The Alzheimer's Disease International Association estimates 36 million people worldwide currently suffer from dementia with this number expected to double every 20 years. With meditation research in its infancy, many medical professionals project this technique to be used as a counter mechanism to fight dementia after its onset as well as as a preventative measure for those who are at risk.

A study was conducted at the University of Pennsylvania, of 50 subjects ranging from 52 and 72 who have a history of memory complications or a diagnosis of cognitive impairment (Newberg, A. B., et al. 2006). One group was instructed to practice a 12-minute guided meditation every day for eight weeks while the others were said to have been placed on a waiting list. After examination of cognitive tests and a single photon emission computed tomography (SPECT) scan, measuring cerebral blood flow through brain imaging, it was found that those who practiced meditation showed significantly higher activity in the posterior cingulate gyrus region, associated with learning and memory, and the first area of the brain to decline in function in the onset of Alzheimer's disease. All participants who underwent the meditation regimen saw at least some statistically significant improvement in cognitive ability–

more specifically improvements in verbal memory, executive function, and attention. Principle Investigator Andrew Newberg M.D., assistant professor of radiology at the University of Pennsylvania School of Medicine, added

> For the first time, we are seeing scientific evidence that meditation enables the brain to actually strengthen itself, and battle the processes working to weaken it.

More evidence suggests that meditation can even be used as a preventative mechanism before neurodegeneration.

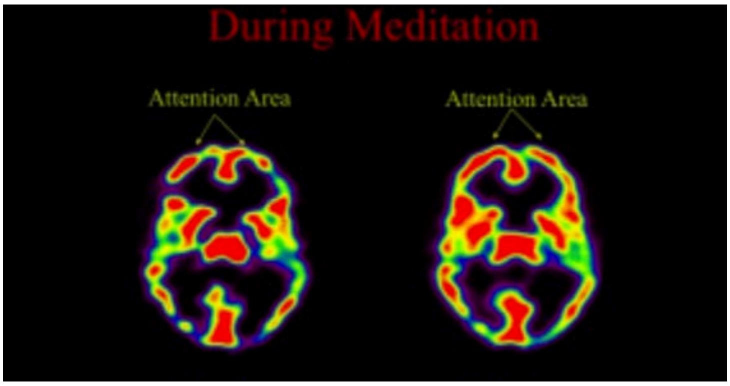


Figure 2. Side by side MRI comparison of brain during meditation with attentional area on the frontal lobe (Newberg et al., 2006)

In 2005, Dr. Lazar conducted a study with 35 participants: 15 control subjects representing the average person and 20 practitioners of Vipassana, a form of mindfulness meditation typically taught over 10 days with 10 hours of daily practice and minimal resources.. The objective was to compare the speed of neurodegeneration between the control and meditation practitioners through observation of cortical thickness in 2 of the commonly at-risk areas of the brain–the right insula and right frontal cortex. Cortical thickness was measured at thousands of points in the vicinity of the area, between inner white and gray matter as well as outer gray and cerebrospinal fluid boundaries. Analysis revealed a significant interaction between group type and age in the right frontal brain region. While participants in the control group demonstrated marked age-related cortical thinning ($r = -0.76$; $P = 0.001$), the study did not find evidence of significant differences in cortical thickness between older and younger meditation practitioners ($r = -0.05$; $P = 0.83$). However, this result does not confirm the absence of a difference and may require further investigation with larger samples. Researchers noted that the average cortical thickness in meditation practitioners aged 40-50 was comparable to that of individuals aged 20-30, including both meditators and controls. This suggests that meditation may help preserve cortical integrity and mitigate age-related neural degeneration and that practicing meditation regularly can slow the rate of natural neurodegeneration at risk locusts and preserve these areas for longer periods of time. Damage to these areas creates at-risk individuals for other neurocognitive diseases that commonly come with age such as Alzheimer's Disease and dementia (Lazar et al, 2005).

Assorted Meditations, with Assorted Effects

Being an ancient technique, meditation has evolved into hundreds of practices worldwide today, some being connected to religion while others being used as relaxation methods in schools. As a result, each form of meditation serves a different purpose and many times a different outcome and physiological change in the body, with some being more effective than others. The most common forms of meditation include Zen, which focuses on seated meditation and breath awareness; Vipassana, a practice rooted in observing sensations and thoughts to develop insight; mindfulness, where attention is brought to the present moment without judgment; Loving-kindness, which involves cultivating compassion and positive feelings toward oneself and others; and Transcendental Meditation, a technique using a mantra to reach a deep state of relaxation and awareness. As a result, no definition of meditation is truly all-encompassing. The scientific community began to study the differences to help categorize but their findings have revealed the benefits of each.

A study published in The Journal of Personality and Social Psychology, aimed to find if there was a difference in effectiveness between the three in terms of retaining cognitive ability, the findings were aimed to determine which would be most beneficial to those suffering with neurocognitive diseases.The study involved 73 seniors with an average age of 81, who were randomly divided into four groups. Three groups practiced different meditation techniques for 12 weeks while the fourth group served as the control group. The techniques included transcendental meditation, mindfulness meditation, and a breath work relaxation program which were to be practiced twice a day for 20 minutes for 12 weeks. The study included a variety of tests to get a cohesive idea of the specific effects each meditation technique was offering, including the Stroops test for cognitive flexibility, associate learning subtest for memory, and word fluency scale/overlearned verbal task for verbal fluency of elderly. The tests were administered before and after the meditation regimen, after 18 months, and after 3 years. The results suggest a strong improvement in measured variables in the group of subjects using transcendental meditation, followed by mindfulness. Worse results on memory tests were shown in the control group and in the group with the relaxation program. In addition, testing after 3 years revealed 100% maintained effects of enhanced cognitive ability in persons using transcendental meditation and 87.5% in those within the mindfulness program. Other groups had lower scores (65 and 75%) (Alexander et al 1989).

Conclusion

Meditation is emerging as a promising tool with profound effects on the brain and body. Research has shown its ability to enhance neuroplasticity, reduce chronic stress, and improve cognitive functioning, all of which are critical in preventing or slowing neurodegenerative diseases such as Alzheimer's and Parkinson's (Lavretsky et al., 2015). By decreasing brain inflammation, promoting healthy neural connections, and reducing oxidative stress, meditation may serve as a non-invasive, cost-effective preventive strategy for these and other age-related conditions (Creswell et al., 2012).

Ongoing studies are focusing on how meditation can influence specific biomarkers associated with neurodegeneration, such as amyloid-beta plaques in Alzheimer's or alpha-synuclein in Parkinson's. Other research is delving into the role of meditation in enhancing mitochondrial health (Epel et al., 2009) and reducing systemic inflammation—factors linked to many chronic diseases, including cardiovascular issues, diabetes, and autoimmune conditions. Advances in neuroimaging and biomarker analysis are helping researchers identify the precise neural pathways and physiological changes meditation induces, offering insights into personalized prevention strategies.

As this field expands, meditation may become an integral part of preventive healthcare. It holds promise not only for delaying the onset of neurodegenerative diseases but also for improving overall health, reducing the prevalence of chronic conditions, and fostering resilience against the challenges of aging. By combining ancient wisdom with modern science, meditation could redefine our approach to health and disease prevention.

References

1. Alexander, C. N., Robinson, P., & Rainforth, M. V. (1989). A model for maintaining cognitive ability in elderly individuals through meditation. Journal of Personality and Social Psychology, 56(4), 581-590.

2. Chaix, R., Gassama, M., Messaoudi, M., & Brigitte, H. (2017). Meditation induces structural changes in the brain: A study of its role in neurogenesis and its potential implications. Frontiers in Psychology, 8, 430.

3. Creswell, J. D., Pacilio, L. E., Doll, R., & Saron, C. D. (2012). Mindfulness meditation and reduction of psychological distress in cancer patients: A meta-analysis. *Journal of Psychosocial Oncology, 30*(3), 256-276.

4. Davidson, R. J., & McEwen, B. S. (2012). Social influences on neuroplasticity: Stress and interventions to promote well-being. Nature Neuroscience, 15(5), 689-695.

5. Epel, E. S., Blackburn, E. H., Lin, J., Dhabhar, F. S., Adler, N. E., Morrow, J. D., & Cawthon, R. M. (2004). Accelerated telomere shortening in response to life stress. Proceedings of the National Academy of Sciences, 101(49), 17312-17315.

6. Hölzel, B. K., Carmody, J., Vangel, M., Congleton, C., Yerramsetti, S. M., & Gard, T. (2011). Mindfulness practice leads to increases in regional brain gray matter density. Psychiatry Research: Neuroimaging, 191(1), 36-43.

7. Lazar, S. W., Hölzel, B. K., & Houghton, D. (2005). Meditation experience is associated with increased cortical thickness. NeuroReport, 16(17), 1893-1897.

8. Lavretsky, H., O'Hara, R., & Epel, E. (2015). Meditation, aging, and neurodegenerative disease. Journal of Clinical Psychology, 71(10), 943-953.

9. Newberg, A., Alavi, A., Baime, M., Pourdehnad, M., Santanna, J., & d'Aquili, E. (2001). The measurement of regional cerebral blood flow during the complex cognitive task of meditation. Psychiatry Research: Neuroimaging, 106(2), 113-122.

10. Newberg, A. B., Iversen, J. P., & Jones, J. (2006). Meditation and neuroimaging: A study on brain function in long-term practitioners. Journal of Cognitive Enhancement, 9(4), 373-388.

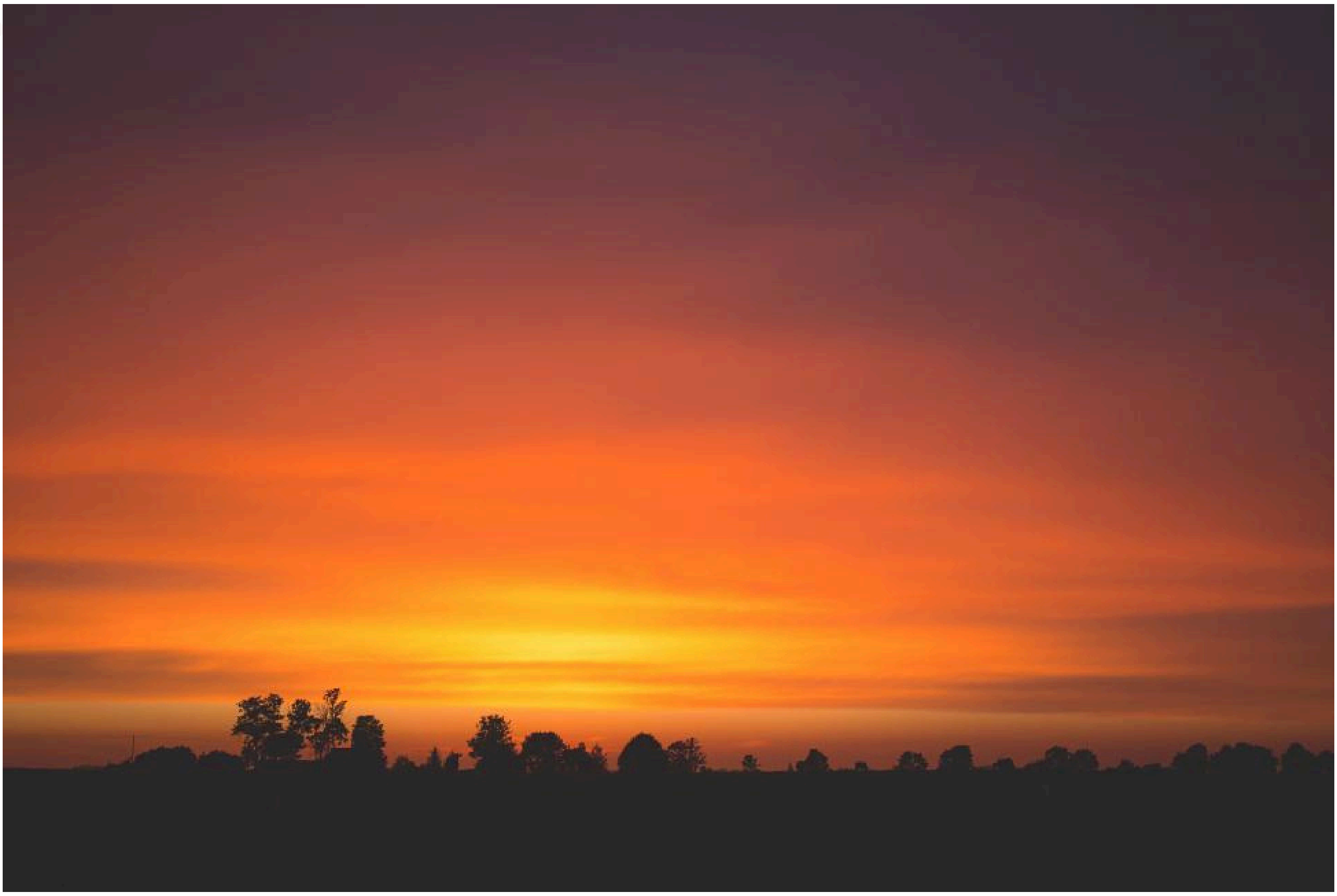
11. Rodriguez-Larios, J., Bracho Montes de Oca, E. A., & Alaerts, K. (2021). The EEG spectral properties of meditation and mind wandering differ between experienced meditators and novices. *NeuroImage*, 245. https://doi.org/10.1016/j.neuroimage.2021.118669

12. Sharma, V. K., & Sood, N. (2015). Neuroplasticity and meditation: A review of the literature. Annals of the New York Academy of Sciences, 1369, 145-156. https://doi.org/10.1111/nyas.12217

13. Taren, A. A., Creswell, J. D., & Matthis, M. (2013). Mindfulness meditation training and the amygdala: Effects on emotion regulation and stress resilience. Social Cognitive and Affective Neuroscience, 8(2), 80-88.

14. Thau L, Gandhi J, Sharma S. Physiology, Cortisol. [Updated 2023 Aug 28]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK538239/

15. Yang, J. L. (2019). Long-term meditation practice leads to neuroplasticity in regions associated with attention and emotion regulation. Frontiers in Psychology, 10, 1930. 16. Zeidan, F., Johnson, S. K., Diamond, B. J., & David, Z. (2011). Mindfulness meditation improves cognition: Evidence of brief mental training. Consciousness and Cognition, 19(2), 201-212.

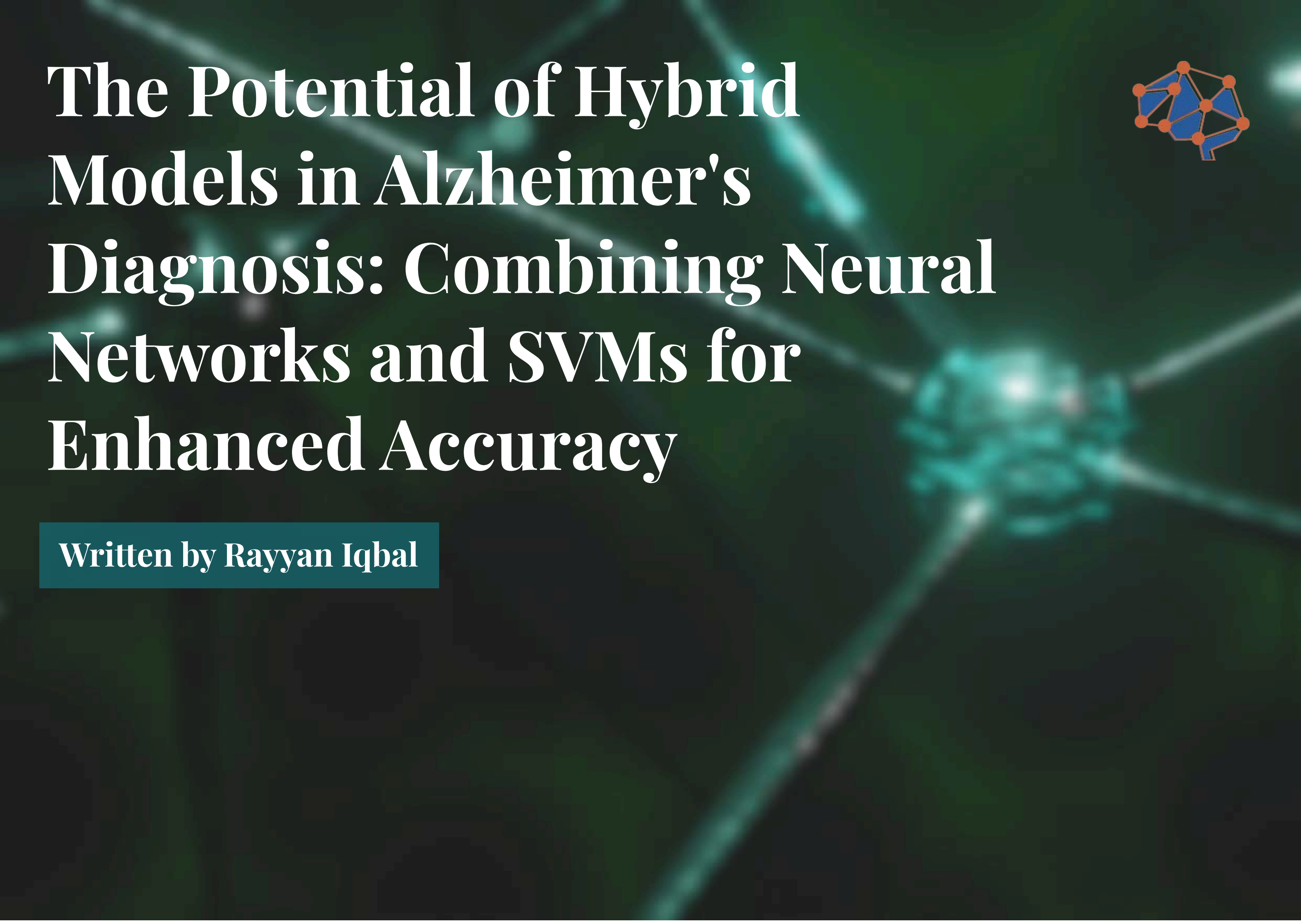


About the Author

Anika Chandola is a Freshman Majoring in MCB with a minor in Psychology and Chemistry on the Pre-Med track. She is currently working in Bagchi Lab researching the environmental impact on reproductive health and volunteered at UChicago's Phlebotomy Clinic. She is also a member of the Gamma Phi Beta Sorority working alongside Girls on the Run. In her freetime she enjoys fashion, swimming, pageants and playing the piano. She hopes to become more involved in the neuroscience field and learn more about this diverse community.

The Potential of Hybrid Models in Alzheimer's Diagnosis: Combining Neural Networks and SVMs for Enhanced Accuracy

Written by Rayyan Iqbal



Introduction

Alzheimer's Disease (AD) affects over 55 million people worldwide, posing a major challenge for healthcare systems as populations continue to age. As a progressive neurodegenerative disorder, AD leads to severe cognitive decline, memory loss, and a profound reduction in quality of life, ultimately resulting in death. The mortality rate for AD approaches 100%, with patients typically living only 3 to 11 years after diagnosis, underscoring the critical importance of early detection (Alzheimer's Stages: How the Disease Progresses, n.d.). Despite extensive research, early diagnosis of AD remains difficult.

The need for early detection is clear when observing cognitive tests like clock-drawing exercises. The differences between healthy individuals and those in late-stage AD are striking; however, distinguishing early-stage AD from normal aging is much harder, as seen in the clock drawings, in which there isn't as profound of a difference in the quality of the clock drawn between normal and early AD individuals (Mattson 2014). Additionally, brain degeneration progresses significantly as AD advances, as shown in the brain degeneration image (Maha 2023). While late-stage AD presents notable structural changes, the brain deterioration in early stages is minimal, making it challenging to identify AD before symptoms become severe.

Traditional diagnostic methods—including clinical assessments, cognitive tests, and neuroimaging—often struggle to detect AD at these early stages, delaying the possibility of effective interventions. Recent advancements in machine learning offer new hope. Neural networks (NNs), particularly convolutional neural networks (CNNs), excel at extracting complex patterns from large datasets, such as MRI or PET scans, allowing for a more nuanced analysis of brain images (Taherdoost 2023). Support vector machines (SVMs), powerful classification tools, complement this process by distinguishing between healthy individuals and those with various degrees of cognitive impairment (Ahmadi et al., 2024).

While each model has shown promise independently, hybrid models that combine the strengths of CNNs and SVMs could further improve diagnostic accuracy. Hybrid models leverage CNNs' ability to identify intricate patterns and SVMs' adeptness at classification, potentially making early detection achievable (Li 2023). However, research in this area is still limited, with few large-scale studies validating these methods. Before these hybrid models can be integrated into clinical practice, more robust data and validation are necessary to ensure their reliability. While the potential of these hybrid models is promising, caution is urged in their application until further research solidifies their effectiveness.

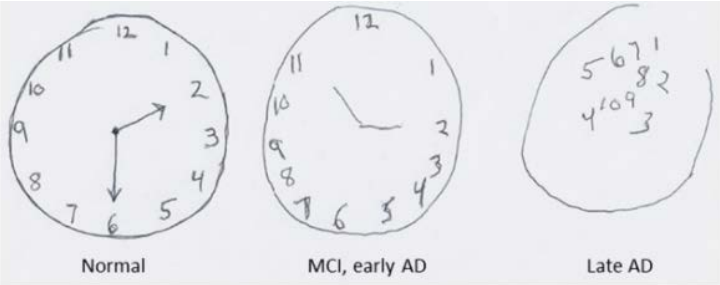


Figure 1. Clock drawings showing cognitive decline: Normal (accurate), Early AD (slightly distorted), Late AD (severely disorganized). (Linus Health)

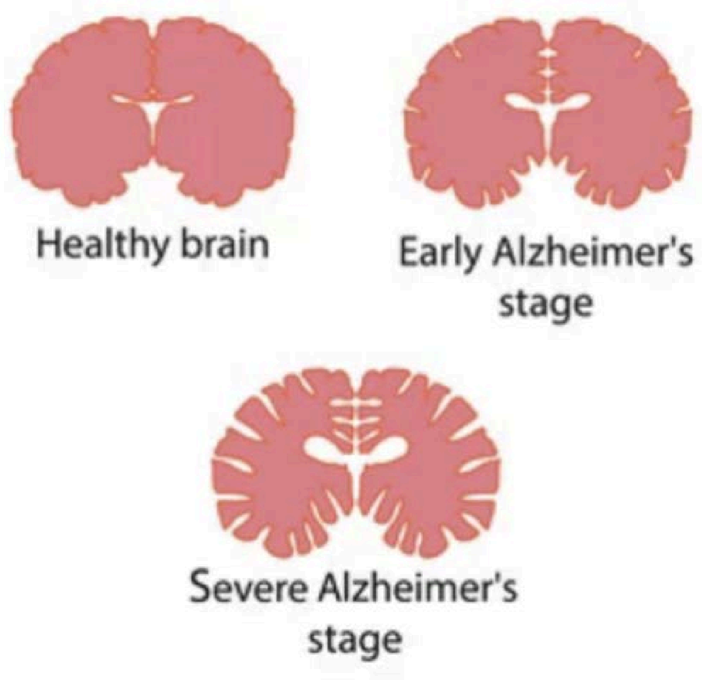


Figure 2. Brain atrophy progression: Healthy, Early Alzheimer's (mild shrinkage), Severe Alzheimer's (significant shrinkage). (First Choice Neurology 2019).

Neural Networks: Identifying Early-Stage Alzheimer's in Neuroimaging

Neural networks, especially convolutional neural networks (CNNs), have proven highly effective at analyzing neuroimaging data like MRI or PET scans, which is essential for diagnosing Alzheimer's Disease in its early stages. Neural networks are inspired by the human brain's structure, consisting of interconnected layers of "neurons" that learn from data by recognizing patterns and relationships. When given input data (such as numbers, images, or text), the network adjusts the connections between these neurons to improve its understanding. This allows neural networks to perform tasks such as predicting outcomes, classifying objects, and identifying trends (Taherdoost 2023).

CNNs are a specialized type of neural network designed specifically for image data. They use a process called "convolution," where small filters scan sections of an image to identify important features like edges, shapes, or textures. Rather than analyzing the entire image at once, CNNs focus on small regions at a time, making them exceptionally good at interpreting visual information. In simple terms, while a standard neural network might act as a general "decision-maker," CNNs operate more like a team of "spotters," with each focusing on a different part of the image to capture crucial details (e.g., one "spotter" might identify the nose, another the eyes). This approach allows CNNs to excel at tasks such as recognizing objects in photos or detecting abnormalities in medical images.

CNNs can achieve remarkable accuracy in identifying early signs of Alzheimer's. In one study using a dataset of healthy individuals, those with early Alzheimer's, and those with late-stage Alzheimer's, a CNN model called ResNet-18 achieved an impressive 96.85% accuracy in distinguishing between different stages of the disease based on MRI and PET scans (Odusami et al., 2021). This performance far exceeds conventional diagnostic methods, such as physician evaluations of PET scans, which typically achieve about 85% accuracy, according to the Texas Department of State Health Services (Texas DSHS, 2021).

However, CNNs have limitations. One major drawback is their "black-box" nature, meaning they generate results without revealing how they reached those conclusions. This lack of transparency can be problematic in clinical settings, where doctors need to understand the reasoning behind a diagnosis to justify treatment decisions. Without insight into how CNNs make their determinations, it becomes challenging for physicians to fully trust and adopt these models in practice, despite their accuracy (Patil et al., 2022).

SVMs: Precise Classification but Limited in Complex Data

While CNNs are excellent for identifying features in images, support vector machines (SVMs) are highly effective for classifying data once those features are identified. The image above illustrates how an SVM functions: two sets of data points, shown as green and red dots, represent two different classes the SVM aims to separate. The axes, labeled x1 and x2, represent a two-dimensional feature space where the data points are plotted. The blue line running diagonally across the plot is the decision boundary, which separates the two classes. This boundary isn't just any line—it is chosen to maximize the distance between itself and the nearest data points from each class, a concept known as the "margin." One of the points closest to the boundary, called a "support vector" (represented by the purple dot), plays a crucial role in defining this margin (Tan 2020).

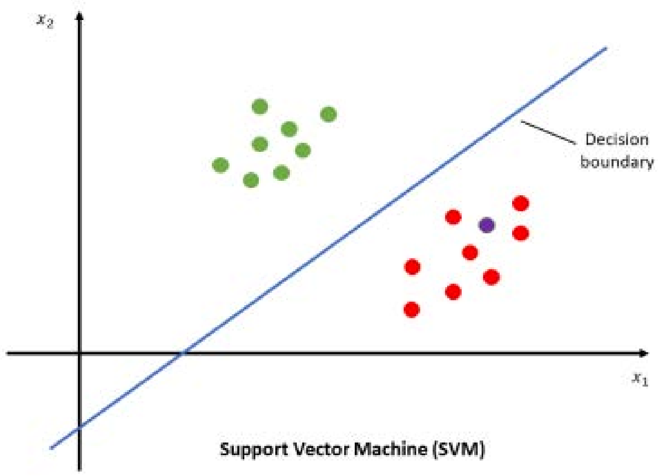


Figure 3. Support Vector Machine (SVM) diagram showing a decision boundary separating two classes. (Tan 2020).

SVMs have demonstrated reliable performance in classifying patients by analyzing MRI data, with their highest accuracy reaching 89%. They excel in identifying key features like gray matter volume and brain asymmetry to distinguish between control groups, early mild cognitive impairment (eMCI), late mild cognitive impairment (lMCI), and Alzheimer's disease stages. In one approach, researchers used hippocampal volume—a primary indicator of Alzheimer's—as the main feature, which contributed to the high accuracy achieved in their study (Ahmadi et al., 2024).

Despite their strengths, SVMs face challenges, particularly with high-dimensional data, where each data point has numerous characteristics or "dimensions." In such cases, it becomes harder for SVMs to identify clear patterns or find a meaningful decision boundary. Without careful feature selection and model tuning, SVMs may struggle to classify complex data accurately, leading to overfitting. Overfitting occurs when a model performs well on training data but poorly on new, unseen data, reducing its real-world effectiveness (Ameen et al., 2024).

Hybrid Models: Combining Neural Networks and SVMs for Enhanced Accuracy

In recent years, hybrid models that combine convolutional neural networks (CNNs) and support vector machines (SVMs) have gained attention in Alzheimer's disease diagnosis, offering a more comprehensive approach to early detection and classification (Li 2023). By integrating CNNs and SVMs, these models provide a robust framework to handle the complexity of high-dimensional neuroimaging data, which is essential for identifying subtle early-stage markers of Alzheimer's.

Hybrid models leverage the unique strengths of CNNs and SVMs. CNNs are particularly skilled at extracting features from complex neuroimaging data, such as magnetic resonance imaging (MRI) and positron emission tomography (PET) scans. These models can detect early neurodegenerative changes, including hippocampal atrophy, cortical thinning, and ventricular enlargement—key biomarkers for diagnosing Alzheimer's at its early stages (Oostven 2021). However, while CNNs excel at identifying these features, they often lack the precision needed for effective classification. This is where SVMs become valuable, as they create precise boundaries that distinguish different cognitive states, such as healthy controls, patients with mild cognitive impairment (MCI), and those with Alzheimer's disease (Ahmadi et al., 2024).

A study by Al Subaie et al. demonstrated the effectiveness of a CNN-SVM hybrid model applied to neuroimaging data, showing the model's ability to distinguish between MCI patients likely to progress to Alzheimer's and those who are not. This research highlighted the advantage of combining CNNs' feature extraction capabilities with SVMs' classification strength. The hybrid model achieved a higher accuracy of 98.20%, compared to 91.70% for models relying solely on CNNs or SVMs. The model was particularly successful in detecting subtle brain changes indicative of MCI conversion, which is essential for timely intervention (Al Subaie et al., 2024).

Basheera et al. explored hybrid deep learning models using multimodal neuroimaging data, combining MRI and PET scans (Basheera et al., 2019). In this approach, the CNN component analyzed the raw imaging data to extract patterns of brain degeneration, which the SVM then classified into diagnostic categories like non-demented, MCI, and AD. This hybrid method demonstrated superior diagnostic accuracy, especially in differentiating between early MCI and advanced cognitive decline stages. The results showed that the CNN-SVM model outperformed traditional diagnostic methods and standalone machine-learning models in terms of accuracy, sensitivity, and specificity, achieving 90.47% accuracy, 86.66% recall, and 92.59% precision (Basheera et al., 2019).

Both studies emphasize the potential of hybrid models to bridge the gap between feature extraction and classification, especially when dealing with high-dimensional neuroimaging data. By allowing CNNs to identify complex biomarkers and enabling SVMs to classify these features accurately, hybrid models offer a more nuanced approach to diagnosing Alzheimer's disease. This combination captures subtle brain patterns that conventional methods might miss, which is crucial for early detection. With earlier intervention, hybrid models could lead to more accurate diagnoses and better treatment options, potentially improving patient outcomes.

Considerations and Future Directions: Evaluating the Potential of Hybrid Models for Clinical Adoption

While hybrid models combining neural networks (NNs) and support vector machines (SVMs) show significant potential in improving the accuracy of Alzheimer's diagnosis, several challenges currently limit their adoption in clinical practice.

One major challenge is the complexity and quality of data required for these models to perform effectively. CNNs, a critical component of hybrid models, rely heavily on neuroimaging data, which is often difficult and costly to obtain at the scale needed for robust machine learning. Studies, including those focusing on multimodal approaches, have highlighted that even well-curated datasets like the Alzheimer's Disease Neuroimaging Initiative (ADNI) are often insufficiently large or diverse to ensure the generalizability of CNNs across various populations. Additionally, collecting such data is complicated by privacy concerns, which can further limit the availability of comprehensive datasets (Ismail et al., 2022).

Another significant challenge is the interpretability of hybrid models. Neural networks, especially CNNs, often function as "black boxes," producing diagnostic results without revealing how they arrived at those conclusions. This lack of transparency makes it difficult for healthcare professionals to understand and trust the model's rationale, particularly when making critical patient care decisions. While efforts like Explainable AI have aimed to make machine learning models more interpretable, the need for transparent decision-making remains a major barrier to adoption in clinical settings (Prijs et al., 2022).

In addition, hybrid models are prone to overfitting—a problem where the model performs well on training data but poorly on new, unseen data. This is especially relevant for models that incorporate both SVMs and CNNs, as SVMs can struggle with high-dimensional data without careful feature selection, and CNNs require extensive computational resources and large datasets to avoid overfitting. If not properly managed, overfitting can undermine the model's real-world effectiveness and limit its reliability in clinical settings (Ying 2019).

Overall, while hybrid models hold great promise, they are not yet ready for widespread clinical adoption. More research is needed to validate their performance across larger, more diverse datasets and to address issues related to interpretability and scalability. Addressing these challenges will be essential for realizing the full potential of hybrid models in Alzheimer's diagnosis and ensuring they can be safely and effectively integrated into everyday clinical practice (Wang et al., 2024).

Conclusion

The integration of neural networks and support vector machines represents a promising approach to improving early Alzheimer's diagnosis. Neural networks, particularly convolutional neural networks (CNNs), excel at identifying subtle patterns in neuroimaging data, while SVMs provide precise classification capabilities. By combining these techniques in hybrid models, diagnostic accuracy can be significantly enhanced, potentially enabling earlier intervention and improved patient outcomes (Oostven et. al., 2021).

However, despite these advancements, the field remains under-researched. The studies conducted so far, while encouraging, are not yet sufficient to support the widespread adoption of hybrid models in clinical practice. More research is necessary to validate these models on larger and more diverse datasets and to address their limitations, particularly in terms of neural network interpretability and the extensive feature selection required for SVMs.

As machine learning continues to evolve, hybrid models combining neural networks and SVMs may ultimately transform Alzheimer's diagnosis, enabling more accurate and timely detection. For now, however, these methods should be approached with caution, as further rigorous and repetitive studies are needed to confirm their reliability in clinical settings. With continued research, these models could one day offer a breakthrough in diagnosing and managing Alzheimer's disease.

References

1. Ahmadi, M., Javaheri, D., Khajavi, M., Danesh, K., & Hur, J. (2024). A deeply supervised adaptable neural network for diagnosis and classification of Alzheimer's severity using multitask feature extraction. PLoS ONE, 19(3), e0297996. https://doi.org/10.1371/journal.pone.0297996

2. Ameen, T. B., Kashif, S. N., Abbas, S. M. I., Babar, K., Ali, S. M. S., & Raheem, A. (2024). Unraveling Alzheimer's: the promise of aducanumab, lecanemab, and donanemab. The Egyptian Journal of Neurology Psychiatry and Neurosurgery, 60(1). https://doi.org/10.1186/s41983-024-00845-5

3. Alsubaie, M. G., Luo, S., & Shaukat, K. (2024). Alzheimer's Disease Detection Using Deep Learning on Neuroimaging: A Systematic review. Machine Learning and Knowledge Extraction, 6(1), 464–505. https://doi.org/10.3390/make6010024

4. Basheera, S., & Ram, M. S. S. (2019). Convolution neural network–based Alzheimer's disease classification using hybrid enhanced independent component analysis based segmented gray matter of T2 weighted magnetic resonance imaging with clinical valuation. Alzheimer's & Dementia: Translational Research & Clinical Interventions, 5(1), 974–986. https://doi.org/10.1016/j.trci.2019.10.001

5. Ismail, W. N., PP, F. R., & Ali, M. A. S. (2022). MULTFORAD: Multimodal MRI neuroimaging for Alzheimer's disease detection based on a 3D convolution model. Electronics, 11(23), 3893. https://doi.org/10.3390/electronics11233893

6. Li, M., Jiang, Y., Zhang, Y., & Zhu, H. (2023). Medical image analysis using deep learning algorithms. Frontiers in Public Health, 11, 1273253. https://doi.org/10.3389/fpubh.2023.1273253

7. Odusami, M., Maskeliūnas, R., Damaševičius, R., & Krilavičius, T. (2021). Analysis of Features of Alzheimer's Disease: Detection of Early Stage from Functional Brain Changes in Magnetic Resonance Images Using a Finetuned ResNet18 Network. Diagnostics, 11(6), 1071. https://doi.org/10.3390/diagnostics11061071

8. Patil, V., Madgi, M., & Kiran, A. (2022). Early prediction of Alzheimer's disease using convolutional neural network: a review. The Egyptian Journal of Neurology Psychiatry and Neurosurgery, 58(1). https://doi.org/10.1186/s41983-022-00571-w

9. Taherdoost, H. (2023). Deep Learning and Neural Networks: Decision-Making Implications. Symmetry, 15(9), 1723. https://doi.org/10.3390/sym15091723

10. Van Oostveen, W. M., & De Lange, E. C. M. (2021). Imaging Techniques in Alzheimer's Disease: A review of applications in early diagnosis and longitudinal monitoring. International Journal of Molecular Sciences, 22(4), 2110. https://doi.org/10.3390/ijms22042110

11. Wang, Y., Gao, R., Wei, T., Johnston, L., Yuan, X., Zhang, Y., & Yu, Z. (2024). Predicting long-term progression of Alzheimer's disease using a multimodal deep learning model incorporating interaction effects. Journal of Translational Medicine, 22(1). https://doi.org/10.1186/s12967-024-05025-w

12. Ying, X. (2019). An Overview of Overfitting and its Solutions. Journal of Physics: Conference Series, 1168, 022022. https://doi.org/10.1088/1742-6596/1168/2/022022

Images:

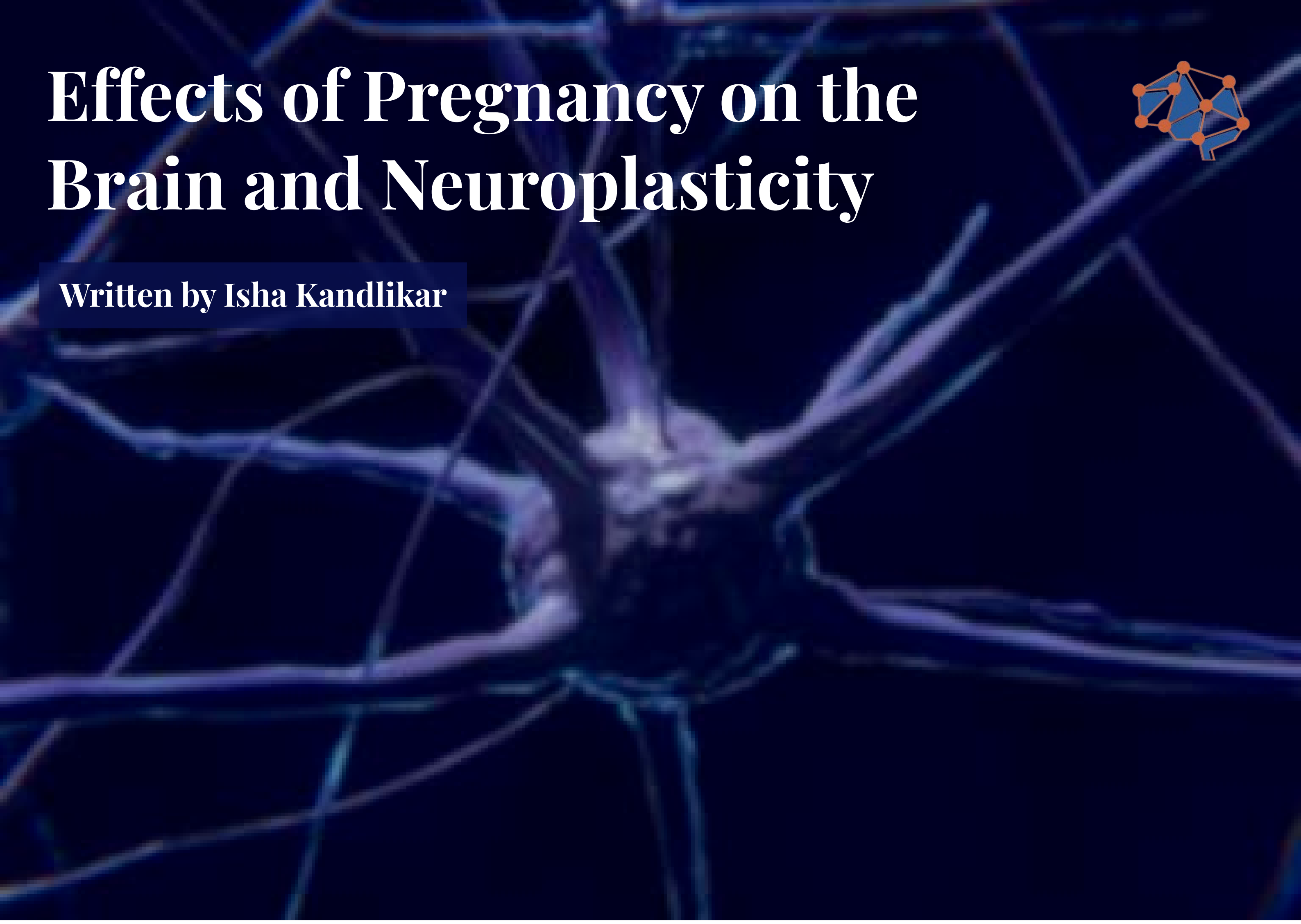
1. First Choice Neurology. (2019). June is Alzheimer's and Brain Awareness Month. *First Choice Neurology.* Retrieved February 18, 2025, from https://www.fcneurology.net/june-is-alzheimers-and-brain-awareness-month/

2. Linus Health. (n.d.). History of the Clock Drawing Test and the Linus Health Platform. *Linus Health.* Retrieved February 18, 2025, from https://linushealth.com/history-of-clock-drawing-test-and-dctclock

3. Tan, A. (2020). Support Vector Machine. *Anthony's Blogs.* Retrieved February 18, 2025, from https://anthony-tan.com/Discriminant-Functions-and-Decision-Boundary/

About the Author

Rayyan Iqbal is a sophomore at the University of Illinois, majoring in Chemistry. He is currently conducting research in the Physical Activity and Neurocognitive Health Lab, where he studies the impact of physical behaviors—such as physical activity and sedentary time—on brain health. Beyond his research, Rayyan is actively involved in REACT, an outreach program that brings science to life for young students in the Champaign-Urbana area.



Effects of Pregnancy on the Brain and Neuroplasticity

Written by Isha Kandlikar

Introduction to Pregnancy-Induced Neuroplasticity

The brain constantly reshapes itself and adapts throughout pregnancy, utilizing a process called neuroplasticity. In this process, the brain undergoes structural changes and reorganization in response to injury, environmental changes, learning, and a variety of other experiences. Pregnancy not only transforms the body but also influences the brain's emotions, maternal instincts, cognition, and overall regulation (Pawluski, Lambert, & Kinsley, 2016). Neuroplasticity, as well as these neurological adaptations, creates long-term shifts in the brain structure and function, which allows the brain to adapt to parenthood and changes in mental state and behavior. Several key neuroplastic changes occur during and after pregnancy, which have implications for maternal cognition and emotional health, highlighting the importance of such changes in maternal and child development.

Hormonal Influences on Maternal Brain Remodeling

During pregnancy, there is a dramatic spike in hormones—primarily estrogen, oxytocin, and progesterone– all of which play a crucial role in shaping the mother's brain for the long-term experiences following pregnancy. According to the NIH, progesterone, which regulates the menstrual cycle and maintains pregnancy, also reaches peak levels before delivery and has a neuroprotective role that includes reducing stress responses and modulating the limbic system, which regulates emotions (Cable, 2023). However, after the mother gives birth, progesterone levels rapidly decrease, which leads to the mother's vulnerability towards disorders like postpartum depression. Oxytocin, a hormone that plays a role in social interactions and emotional regulation, steadily increases during pregnancy, then rises dramatically during labor and breastfeeding to encourage uterine contraction and lactation. Oxytocin further remodels the brain through enhancing neural plasticity in the medial preoptic area (MPOA), amygdala, and nucleus accumbens, which are key regions for maternal motivation and emotional bonding (Thul, 2020). This ultimately changes the mother's brain by rewiring and strengthening mother-infant attachment, increasing her sensitivity to infant cues. Not only does oxytocin affect the mother during pregnancy, but it also maintains a lasting effect through breastfeeding, as it promotes emotional resilience and reduces stress by decreasing activity in the hypothalamic-pituitary-adrenal (HPA) axis, which regulates cortisol production (Blankers, 2024). The intricate interplay between oxytocin, progesterone, and estrogen leaves long-term impacts on emotional regulation, cognition, and maternal instincts during and after pregnancy.

Structural Brain Changes: Gray and White Matter

Neuroimaging studies have led researchers to believe that pregnancy induces changes in the regions of the brain that regulate social cognition and emotional regulation (Younis, 2025). One of the most notable findings is gray matter volume reduction in areas such as the prefrontal cortex, medial temporal lobe, and limbic system, primarily in the amygdala and hippocampus. Furthermore, a reduction in gray matter likely reflects a neural pruning process, which shapes maternal instincts and caregiving behaviors. Since these regions are crucial for social cognition and emotional processing, the mother's ability to look after her child and tend to their needs is improved as maternal sensitivity is increased (Snyder, 2017).

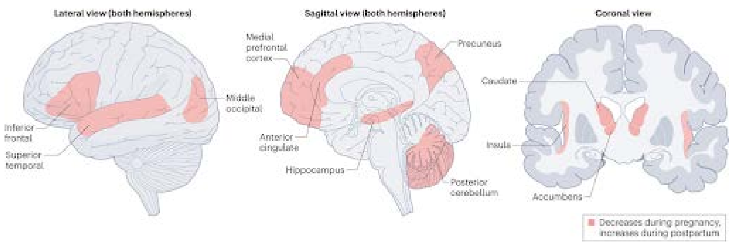


Figure 1. This image illustrates brain regions that decrease in volume during pregnancy and increase postpartum, highlighting areas involved in emotional regulation, cognition, and maternal behavior (Medical Xpress, 2023)

Pregnancy also induces white matter expansion, which speeds up neural transmission and improves connectivity via myelin, a lipid coating of a neuron's axon. White matter tracts facilitate communication between brain regions – this increase in white matter density occurs specifically in pathways that involve cognitive control and emotional regulation. White matter volume is increased in the prefrontal cortex, enhancing cognitive control and decision making (Blankers, 2024). Additionally, there is an observed increase in the cingulum bundle, a white matter tract that connects the prefrontal cortex and limbic system (including the amygdala and hippocampus), playing a role in emotional processing, stress regulation, and maternal motivation (Blankers, 2024). These changes can contribute to lower cortisol levels, which helps the mother remain calm under stress. Increased connectivity in reward pathways may also boost dopamine signaling, enhancing maternal motivation and infant bonding.

Changes in the Default Mode Network and Empathy Processing

In addition to the reduction of gray matter and white matter expansion, pregnancy also alters the functional connectivity within the Default Mode Network (DMN), the inter-regional connections that control empathy, self-reflection, and social processing involving the medial prefrontal cortex, posterior cingulate cortex, and inferior parietal lobule. Research suggests that the reduction of gray matter, particularly in areas associated with theory of mind and social cognition, contributes to changes in DMN connectivity. These changes in the DMN activity highlight a shift in the maternal brain regarding responsiveness towards infant-related stimuli, which continues to reinforce the infant-mother bond (Paternina-Die, Martínez-García, Martín de Blas et al., 2024).

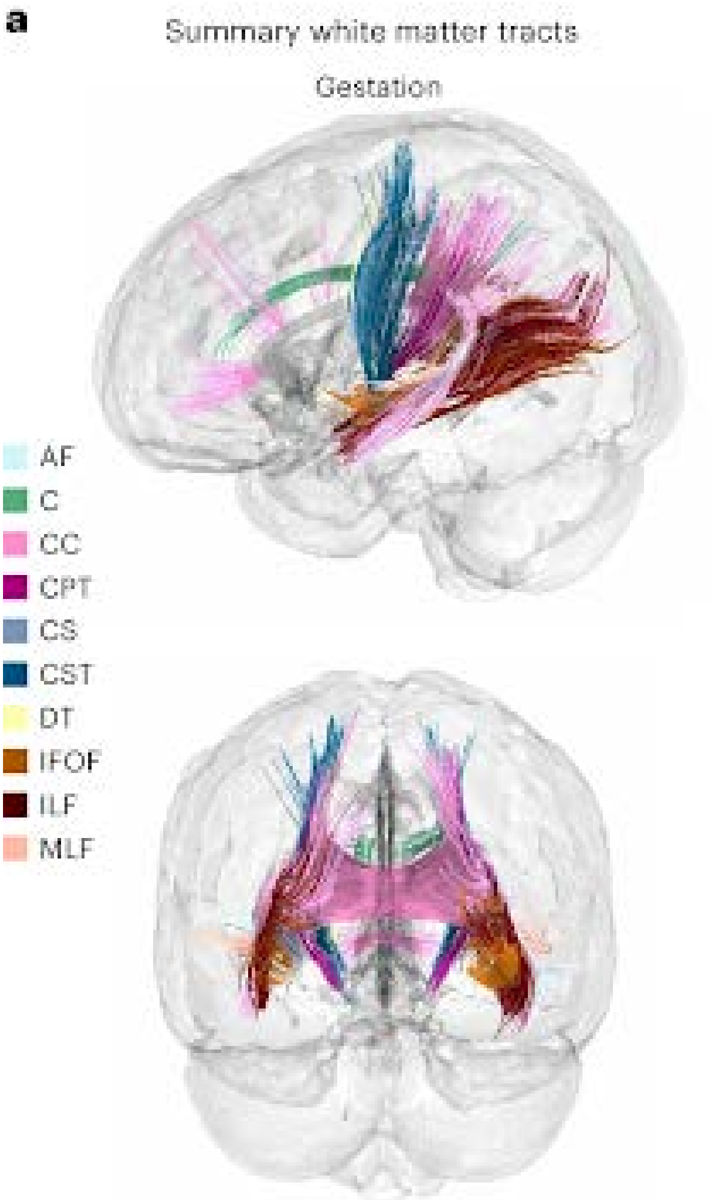


Figure 2. This image shows the distribution of white matter tracts during gestation, emphasizing the enhanced neural connectivity that supports cognitive control, emotional regulation, and maternal behaviors (Pritschet et. al., 2024).

On a much broader scale, pregnancy affects multiple brain structures, including the hippocampus (memory and learning), prefrontal cortex (decision-making and emotional regulation), and amygdala (emotion processing and threat detection). As a whole, these changes enhance maternal responsiveness, improve memory for infant cues, and reduce the fear response to infant stimuli. Improved memory for infant cues allows mothers to better recognize responses such as their baby's facial expression and cries, while reducing fear response to infant stimuli helps the mother stay calm in overwhelming situations. Overall, during pregnancy, the size of the brain is reduced temporarily but returns to its original size 1-2 years postpartum (Kohl, 2019).

Cognitive Shifts and Evolutionary Adaptations

Pregnancy induces cognitive shifts as a result of the changes in neuroplasticity and is commonly referred to as the "mom brain" (Phoenix Health, 2025). This phenomenon is characterized by cognitive fog, forgetfulness, and altered attention during pregnancy and early motherhood. As the size of the brain decreases during pregnancy, it goes through a neural pruning process, which allows it to shift its focus onto infant-related stimuli. For example, mothers may experience a heightened sensitivity to infant cues such as crying, facial expressions, and touch. During pregnancy, many women struggle with focus, verbal fluency, and memory, which is due to the hormonal fluctuations but also to the physical structural changes. The changes and volume reduction of the hippocampus may explain the reduced cognitive flexibility as well as the short term forgetfulness. While these changes may seem negative, they are necessary for evolution and evolutionary mechanisms of the mother during pregnancy and its lasting effects (Phoenix Health, 2025). Neural pruning allows the brain to be more specialized and more efficient by streamlining information deemed necessary to motherhood (Kim, 2010). By restructuring the "mom brain", these changes are optimizing maternal behavior through increases in maternal instincts, emotional regulation, and awareness of infant cues with empathy and vigilance.

While pregnancy is universally known to impact neuroplasticity, affect the hormonal balance, and change the physical structure of the brain, the extent and persistence of such changes vary. Some alterations to the brain and body, such as changes in gray matter volume or hormonal shifts, can persist for months or even years postpartum, influencing long-term maternal behavior and emotional regulation. Mothers experience different changes in neuroplasticity based on genetics, mental health, trauma, and previous life events. Genetic factors contribute to individual differences in terms of hormonal regulation, maternal behavior, brain plasticity, and more. The main variants in genes are related to the oxytocin receptor function (OXTR), dopamine signaling (DRD2), and serotonin regulation (5-HTTLPR), all of which contribute to maternal sensitivity, stress management, and emotional bonding with the infant (Duarte-Guterman, Leuner, & Galea, 2019). Women with genetic predispositions for higher oxytocin receptor sensitivity can experience stronger, more natural maternal instincts and emotional bonding. Mothers with genetic vulnerabilities to dopaminergic dysfunction are more likely to struggle with postpartum mood disorders or attachment difficulties.

Mental Health and Its Influence on Postpartum Brain Adaptation

Furthermore, the mother's mental health and past experiences before pregnancy can significantly impact how her brain adapts to pregnancy and life after. Women with a history of anxiety, depression, or trauma are more likely to exhibit altered neural plasticity in response to pregnancy-related hormonal shifts. This is highlighted in conditions such as pre-existing stress-related dysregulation in the HPA axis, which is correlated to higher susceptibility to postpartum depression (Kim 2010).

The persistence of neuroplasticity postpartum is largely affected by the mother's pre-existing mental health conditions, such as anxiety, depression, or trauma. Research indicates that women with a history of major depressive disorder or anxiety show altered connectivity in maternal brain regions, specifically the prefrontal cortex and amygdala, which both impact emotional responses to infants and motivation for caregiving (Meireles, 2021). Such disruptions can potentially lead to a shortened duration of adaptive neuroplasticity, making it more difficult for these mothers to sustain long-term changes and to support maternal responsiveness and bonding. This not only changes the way the maternal brain adapts, but also the duration of its impact (Meireles, 2021). Conversely, mothers with minimal mental health concerns show stronger connectivity between the amygdala and PFC, allowing for better emotional regulation and lasting positive adaptations. Mothers with past experiences of positive emotional regulation are able to sustain the neuroplasticity changes in the amygdala, reinforcing maternal instincts for longer (Meireles, 2021).

Because pregnancy is a period of significant neuroplastic adaptation, it can induce long-term neurological vulnerabilities. A representative example of this is preeclampsia, a hypertensive disorder that affects approximately 5–8% of pregnancies (Karrar, 2024). Preeclampsia is associated with hypertension, endothelial (relating to blood vessels) dysfunction, and systemic inflammation, which not only pose immediate perinatal risks but can also increase the mother's long-term susceptibility to neurodegenerative diseases such as Alzheimer's disease, vascular dementia, and stroke. This increased vulnerability is hypothesized to stem from persistent endothelial dysfunction and systemic inflammation, which make up cerebrovascular integrity and neuronal function even decades after pregnancy (Logue, 2016). Heightened levels of pro-inflammatory cytokines, such as IL-6, TNF-α, and oxidative stress markers observed in preeclamptic pregnancies can persist postpartum, leading to accelerated neurovascular aging and white matter damage (Friis et al., 2024).

Beyond preeclampsia, there are many neurological vulnerabilities created from the postpartum period itself. Hormonal imbalance and the sudden drop in estrogen and progesterone in the postpartum period can exacerbate neuroinflammatory pathways, increasing the risk of neuropsychiatric disorders and cognitive deficits. Examples of potential neuropsychiatric disorders include postpartum depression, anxiety disorders, and even postpartum psychosis, while examples of cognitive deficits are memory impairments, brain fog, and decreased processing speed (González-Mesa et al., 2020). Additionally, the reduction of the hippocampus postpartum can impair synaptic plasticity specifically in women with pregnancy complications, leading to longer-term cognitive impairment and dysfunction. Some preventative measures to reduce neurodegenerative effects include routine neurological screenings, blood pressure management, and exercise (González-Mesa et al., 2020).

As more than 80% of biological women become mothers by the age of 40 in the USA (Pew Research Center, n.d.), understanding of the effects of pregnancy on neuroplasticity is crucial. Pregnancy induces reshaping of the brain and hormonal changes well past childbirth. These cognitive changes are not always deficits, but adaptations that enhance maternal instincts, emotional regulation, and caregiving behaviors, strengthening the mother-infant bond. Consequently, further neuroscience research is essential in order to improve maternal healthcare, mental health support for mothers, and long-term brain intervention therapies.

Conclusion and Future Directions

Driven by hormonal change and structural remodeling that enhance cognition, maternal behaviors, and emotional regulation, pregnancy overall triggers many neuroplastic changes in the maternal brain. These adaptations and shifts are essential in optimizing the mother's caregiving abilities and responsiveness to the infant. However, many genetic factors, pregnancy complications, and individual mental health struggles influence and affect the persistence of these brain changes. Understanding pregnancy-induced neuroplasticity and continuing research is vital for advancing maternal health care, supporting mental well-being, and addressing long-term neurological risks.

References

1. Barha, C. K., & Galea, L. A. M. (2010). Influence of different estrogens on neuroplasticity and cognition in the hippocampus. Biochimica et Biophysica Acta (BBA) - General Subjects, 1800(10), 1056–1067. https://doi.org/10.1016/j.bbagen.2010.01.006

2. Blankers, S. A., Go, K. A., Surtees, D. C., Splinter, T. F. L., & Galea, L. A. M. (2024). Cognition and neuroplasticity during pregnancy and postpartum. In P. J. Brunton & D. R. Grattan (Eds.), Neuroendocrine regulation of mammalian pregnancy and lactation (Vol. 15). Springer. https://doi.org/10.1007/978-3-031-51138-7_9

3. Cable, J. K., & Grider, M. H. (2023, May 1). Physiology, progesterone. In StatPearls. StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK558960/

4. Duarte-Guterman, P., Leuner, B., & Galea, L. A. M. (2019). The long and short term effects of motherhood on the brain. Frontiers in Neuroendocrinology. https://doi.org/10.1016/j.yfrne.2019.02.004

5. Friis, T., et al. (2024). Gestational Hypertension, Preeclampsia, and Eclampsia and Future Neurological Disorders. JAMA Neurology, 81(4), 4426. https://www.preeclampsia.org/the-news/research/does-preeclampsia-increase-your-risk-for-future-neurological-conditions

6. González-Mesa, E., et al. (2020). The Neuroendocrinological Aspects of Pregnancy and Postpartum Depression. Journal of Clinical Medicine, 9(1), 241. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6992410/

7. Hoekzema, E., van Steenbergen, H., Straathof, M., Servin-Barthet, C., Mutsaerts, H. J. M. M., Pannekoek, J. N., ... & Hommel, B. (2022). Mapping the effects of pregnancy on resting state brain activity, white matter microstructure, neural metabolite concentrations and grey matter architecture. Nature Communications, 13, 6931. https://doi.org/10.1038/s41467-022-33884-8

8. Karrar, S. A., Martingano, D. J., & Hong, P. L. (2024). Preeclampsia. In StatPearls. StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK570611/

9. Kim, P., Leckman, J. F., Mayes, L. C., Feldman, R., Wang, X., & Swain, J. E. (2010). The plasticity of human maternal brain: Longitudinal changes in brain anatomy during the early postpartum period. Behavioral Neuroscience, 124(5), 695–700. https://doi.org/10.1037/a0020884

10. Kohl, S. K., & Leuner, B. (2019). Brain plasticity in pregnancy and the postpartum period: Links to maternal mental health. Frontiers in Neuroendocrinology, 55, 100788. https://doi.org/10.1016/j.yfrne.2019.100788

11. Livingston, G. (2018, January 18). They're waiting longer, but U.S. women today more likely to have children than a decade ago. Pew Research Center. https://www.pewresearch.org/social-trends/2018/01/18/theyre-waiting-longer-but-u-s-women-today-more-likely-to-have-children-than-a-decade-ago/

12. Logue, O. C., George, E. M., & Bidwell, G. L. (2016). Preeclampsia and the brain: Neural control of cardiovascular changes during pregnancy and neurological outcomes of preeclampsia. Clinical Science (London, England: 1979–), 130(16), 1417–1434. https://doi.org/10.1042/CS20160108

13. Medical Xpress. (2023, September 19). Choreography between hormones and the brain key to understanding how women adapt to motherhood. https://medicalxpress.com/news/2023-09-choreography-hormones-brain-key-women.html

14. Meireles, A. L. F., Segabinazi, E., Spindler, C., Gasperini, N. F., dos Santos, A. S., Pochmann, D., Elsner, V. R., & Marcuzzo, S. (2021). Maternal resistance exercise promotes changes in neuroplastic and epigenetic marks of offspring's hippocampus during adult life. Physiology & Behavior, 230, 113306. https://doi.org/10.1016/j.physbeh.2020.113306

15. Paternina-Die, M., Martínez-García, M., Martín de Blas, D., & colleagues. (2024). Women's neuroplasticity during gestation, childbirth and postpartum. Nature Neuroscience, 27, 319–327. https://doi.org/10.1038/s41593-023-01513-2

16. Pawluski, J. L., Lambert, K. G., & Kinsley, C. H. (2016). Neuroplasticity in the maternal hippocampus: Relation to cognition and effects of repeated stress. Hormones and Behavior, 77, 86–97. https://doi.org/10.1016/j.yhbeh.2015.05.004

17. Phoenix Health. (2025). Pregnancy brain: The science behind it & tips to help. Phoenix Health. https://www.joinphoenixhealth.com/resourcecenter/pregnancy-brain-science-and-tips/

18. Pritschet, L., Taylor, C. M., Cossio, D. M., Santander, T., Quintana, D. S., & Jacobs, E. G. (2024). Neuroanatomical changes observed over the course of a human pregnancy. Nature Neuroscience. https://doi.org/10.1038/s41593-024-01741-0

19. Snyder, H. R., Hankin, B. L., Sandman, C. A., Head, K., & Davis, E. P. (2017). Distinct patterns of reduced prefrontal and limbic grey matter volume in childhood general and internalizing psychopathology. Clinical Psychological Science, 5(6), 1001–1013. https://doi.org/10.1177/2167702617714563

20. Thul, T. A., Corwin, E. J., Carlson, N. S., Brennan, P. A., & Young, L. J. (2020). Oxytocin and postpartum depression: A systematic review. Psychoneuroendocrinology, 120, 104793. https://doi.org/10.1016/j.psyneuen.2020.104793

21. Younis, J., Bleibel, M., El Masri, J., Ismail, A., & Abou-Abbas, L. (2025). Exploring the influence of pregnancy on cognitive function in women: A systematic review. BMC Pregnancy and Childbirth, 25(1), 88. https://doi.org/10.1186/s12884-025-07181-3

About the Author

Isha Kandlikar is a rising junior at the UIUC majoring in Molecular and Cellular Biology with minors in Business and Public Health. She is an undergraduate researcher in the Rudolph Lab, where she studies genetic mouse models to explore treatments for psychiatric disorders. Outside the lab, Isha is a marketing and healthcare consultant, a member of the Illini Medical Screening Society, and involved in Alpha Epsilon Delta, a pre-health fraternity on campus. Passionate about neuroscience, she is excited to write for Brain Matters and dive deeper into specific topics within the field.

Adaptive Plasticity of the Colorblind Brain: A Model for Sensory Compensation

Written by Yuliia Kohut

Abstract

Color vision deficiency (CVD) or color blindness results from X-linked recessive genetic mutation that decreases or impairs the expression of cone cell photoreceptors essential for normal color perception. As a result, individuals with color blindness are unable to distinguish certain colors or hues in the same way as individuals with typical color vision. On a molecular level, the most common forms of CVD arise from the absence or malfunction of one type of cone cell in the retina, which reduces sensitivity to specific wavelengths of light. This disruption in normal color processing leads to altered color perception, often making daily visual tasks more challenging. However, the colorblind brain can adapt to these perceptual differences through neural plasticity. Recent neuroscience research indicates that visual cortical areas V2 and V3 are particularly involved in cortical reorganization in individuals with CVD. Additionally, at the cellular level, structures such as rods, intrinsically photosensitive retinal ganglion cells (ipRGCs), and neurons in the lateral geniculate nucleus (LGN) may contribute to compensatory neuroplastic responses to altered visual input. By using current research on the adaptive plasticity of the brain in color blind people, scientists can further the potential of neural training for rehabilitation and therapeutic strategies targeted to treat brain trauma, injuries, or other visual impairments.

Introduction

Around 1 in 12 men and 1 in 200 women are colorblind (Fareed, 2015), yet we rarely consider how they perceive the world around us. Colorblindness is often dismissed as a minor inconvenience, but it could offer a unique opportunity to study how the brain adapts to sensory deficits.

Color blindness, or color vision deficiency (CVD), is a condition characterized by a decreased ability to perceive color differences under normal light conditions and can be genetic or acquired due to trauma. In CVD, cone cells in the eye retina fail to process color information correctly due to malfunctioning or missing opsin proteins (Simunovic, 2009). Depending on the mutation, colorblind individuals may experience anomalous trichromacy (opsins are present but less sensitive), dichromacy (one of the cone types is missing), or monochromacy (all cones are missing or non-functioning). The most common forms, protanomaly (red-weak) and deuteranomaly (green-weak), result in a shifted perception of color, altering how individuals interact with their environment. Current research suggests that the altered photoreceptor function in individuals with color vision deficiency may influence neural processing at the cortical level (Rina, 2024), introducing changes in brain function, especially its plasticity—the brain's ability to 'rewire' itself due to injury or experience (Puderbaugh, 2023). Unlike sudden sensory loss, congenital color blindness is a lifelong deficiency, allowing researchers to explore how the brain adapts to deficiency from an early age (Isherwood, 2020). As a result, studying these adaptations may offer valuable clues for developing therapies to restore vision or improve recovery after brain injury. This review will discuss neural plasticity associated with colorblindness and how current literature suggests these insights can be used to inform broader neuroscience research on therapies for sensory deprivation and sensory repair.

Mechanisms of Color Vision

The physiology of color vision is thought to be the same across all species, yet scientists still have much to uncover. At its core, color vision relies on our brain's ability to analyze the energy and frequency of light scattered by an object, using opsins—light-activated protein receptors—embedded in cell membranes of photoreceptor cells. So, color processing in humans involves two organs: the retina and the brain, specifically the visual cortex in the occipital lobe. Photoreceptor cells, or photoreceptors, are specialized neurons in the eye retina that detect light. There are two major types of photoreceptors in humans: rods that are used for vision in the dark and cones that are used to detect color via opsin proteins, which are, therefore, crucial for understanding color vision deficiency.

Humans have opsin proteins sensitive to short (S; maximally sensitive to blue wavelength light), medium (M; maximally sensitive to green wavelength light), and long (L; maximally sensitive to red wavelength light) wavelengths, resulting in routine trichromatic vision (Isherwood, 2020; Pasmanter). To understand how opsins can detect specific colors of light and transmit signals to the brain, we need to explore the physics of light and the molecular structure of opsin proteins.

According to the electromagnetic spectrum theory in physics, all electromagnetic radiation, which includes light, can be characterized by its wavelength and energy. The visible portion of the electromagnetic spectrum, which we perceive as colors, has wavelengths ranging roughly from 400 to 700 nanometers. Within this spectrum, different wavelengths correspond to different colors; for example, as shown in Figure 1, longer wavelengths appear red, while shorter wavelengths appear violet or blue (Ailioaie, 2020). When referring to S, M, or L cones (or also S, M, or L opsins in other literature), we mean that each type is activated by a specific range of wavelengths with a corresponding energy. This activation induces a conformational change in the opsin protein, triggering a cascade of biochemical reactions that transmit a signal to the brain. As illustrated in Figure 2, opsins act as G-protein coupled receptors. In their signalling conformation opsins can bind to and activate the G protein by catalysing the exchange of GDP (guanosine diphosphate) to GTP (guanosine triphosphate). The GTP-bound Gα subunit dissociates from the Gβγ subunit exposing its active site and binding to its effector, phosphodiesterase. Phosphodiesterase then starts a cascade of reactions that eventually create a hyperpolarization response in the cones. This membrane hyperpolarization in cones modulates the release of neurotransmitters to ganglion cells which form the optic nerve, finally projecting the signal to the brain (Shichida, 2009).

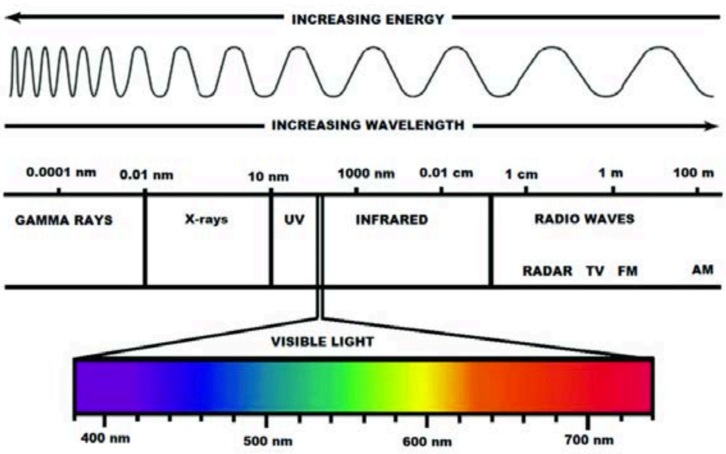


Figure 1. The visible spectrum of light inside the electromagnetic radiation spectrum (Ailioaie, 2020)

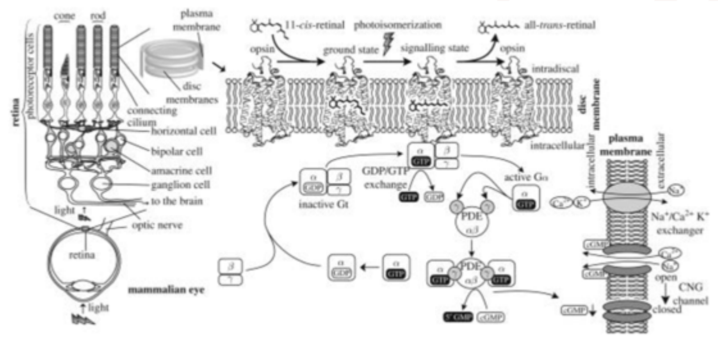


Figure 2. A schematic showing the molecular and physiological mechanism of phototransduction from the retina in mammalian eyes. This schematic uses bovine rhodopsin as an example of opsin protein and its function (Shichida, 2009)

The mechanism of color vision discussed so far is known as trichromatic color theory, which states that our perception of color relies on detecting signal intensities from three types of cones—S, M, and L cones—that correspond to blue, green, and red light. Psychology research also suggests other color vision models, like opponent processing theory, that act in tandem with trichromatic theory to allow us to perceive colors differently (Lee, 2011). Further color encoding (e.g. detecting color hues) depends on neural activity in the visual cortex to compare if a given wavelength, for example, excites M or L cone receptors more (Isherwood, 2020).

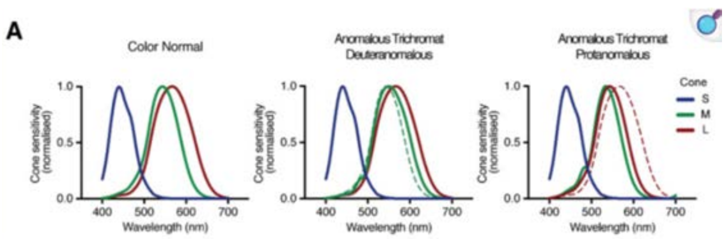


Figure 3. Overlapping sensitivities for different color vision deficiencies (Isherwood, 2020).

Figure 3 from a 2020 review by Isherwood illustrates differences in S, M, and L cone sensitivities in normal color vision as compared to trichromat deuteranomalus or trichromat protanomalus vision. In deuteranomalus vision, the green spectrum(i.e. range of absorbed wavelengths that result in green color) overlaps more with the red spectrum, resulting in green-color weakness. Similarly, the red spectrum shifts closer to the green spectrum sensitivity in protanomalous vision, resulting in a red-color weakness. There are also CVDs where one type of cone is completely missing or non-functioning, resulting in a more drastic change of color vision. When M cones are missing, an individual experiences deuteranopia, leading to the inability to detect green light. Missing L cones leads to protanopia—the inability to detect red light.

Scientists can use computational tools to visualize how individuals with these conditions experience color, see Figure 4 (Wong, 2011). Figure 3 displays a simulation of dichromat percept with decreased L-M light spectrum comparison, assuming this image is a close representation of how protanopes and deuteranopes perceive color.

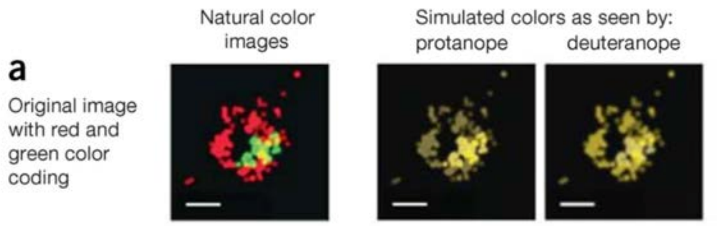


Figure 4. Immunofluorescence image in original color (red and green) and simulated images as seen by protanopes and deuteranopes (Wong, 2011)

Although color blindness might result from a brain injury or eye disease (Cowey, 1997), it is most commonly diagnosed as a congenital condition. The genes that code for opsins are located on the X chromosome. This explains why men are more likely to be color blind than women because mutation on the X chromosome is guaranteed to be expressed in the male population with only one X chromosome copy and not two copies like in females. The genetic designations for the L and M opsin genes are OPN1LW and OPN1MW, respectively. Mutations in these genes, therefore, lead to color vision deficiencies, such as protanopia or deuteranopia, when affecting either OPN1LW or OPN1MW. On a physiological level, mutation in these genes leads to decreased or absent expression of opsin receptors, affecting visual perception of contrast sensitivity, color discrimination, and object recognition. As a result of these genetic changes, colorblind individuals often rely on several post-receptoral adaptations (Isherwood, 2020)—processes in the neural activity of the colorblind brain that help individuals with CVD compensate for receptoral malfunction. Such adaptations are of great interest in neuroscience because they pose questions about mechanisms of neuroplasticity and how the brain adjusts to CVD on the level beyond the eye retina.

Figure 5: Simulated dichromat percept of color (Isherwood, 2020)

Neural Plasticity in Color Vision Deficiencies

Color blindness is a unique "natural experiment" (Isherwood, 2020) to study neural plasticity due to two main reasons. Firstly, CVDs arise from a discrete change on the first step of color vision, which allows us to study brain reorganization as a result of a constant and simple change in informational input like the altered light detection (Isherwood, 2020). Secondly, because each color blind individual spends a lifetime experiencing a defective color vision, which serves as a valuable opportunity to study brain plasticity on timescales much larger than scientists can afford in the lab (Isherwood, 2020).

It is obvious that CVD causes perceptual changes at the retinal level of color vision, but neuroscientists are also interested in studying how deficient light input changes cortical function. Some studies hypothesize that neuroplasticity adapts to CVD through cortical reorganization, where the brain assigns a function to a cortical area that it does not normally have due to altered sensory input.

There is evidence of strong compensation for color losses in anomalous trichromacy via amplification of cortical responses to chromatic contrast in the V1 (primary visual cortex), V2 (secondary visual cortex), and V3 (V1 and V2 signal processing; motion processing) areas of the visual cortex (Tregillus 2021; Huff; Arcaro, 2015). Interestingly, in an fMRI study examining brain activity in colorblind and healthy subjects performing tasks requiring attention fixation on image contrast sensitivity, researchers found that V1 activity was decreased in colorblind individuals, while V2 and V3 activity remained unchanged, meaning that these parts of the visual cortex might play a role in color processing and associations in color blind individuals, see Figure 4 (Tregillus 2021).

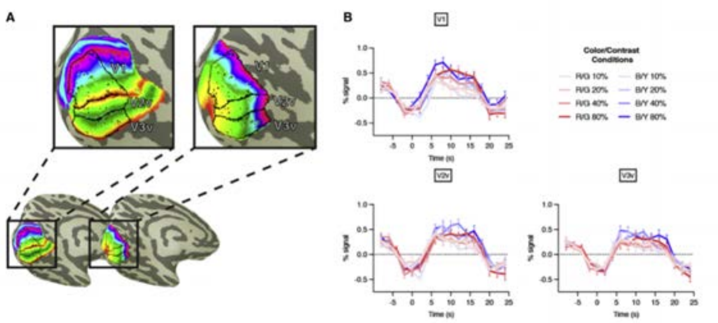


Figure 6. fMRI data on V1, V2, and V3 cortical activity during attention heavy experiments. A. CVD brain activity is on the right of A being compared to normal brain function on the left of A. Retinotopic polar-angle and eccentricity maps (0–9.5°) overlaid on an inflated left hemisphere for V1, V2v, and V3v. The central 0–0.95° (fixation zone) was excluded. B. Elucidation of the results in A heat maps with plots of fMRI signal change. Mean % signal change in V1, V2v, and V3v for one subject, averaged over 6 runs (12 repeats per condition). Each block had 14 s of stimulus followed by an 8 s gray-screen rest. (Tregillus, 2021).

Additional research supports the idea that chromatic adaptation occurs at the retinal level and within central visual pathways. Studies on the McCollough effect (a visual illusion that causes color aftereffects) suggest that some chromatic adaptation mechanisms operate at an early monocular stage (Stromeyer, 1978). However, further experiments indicate that normalization mechanisms extend beyond the retina. Electroretinography (ERG) recordings showed no significant differences in spectral sensitivity before, during, or after chromatic alteration, suggesting that these effects are mediated at a post-receptoral level (Neitz, 2002). Furthermore, monocular chromatic alteration experiments demonstrated interocular transfer of color perception shifts, supporting that chromatic adaptation occurs within central visual pathways at a postsynaptic locus where chromatic information from both eyes has already been integrated (Neitz, 2002). Other studies also suggest that the cortex shows much more plasticity related to color-contrast adaptation compared to the lateral geniculate nucleus (LGN), which is a part of the thalamus that relays information from the retina to the visual cortex or retinal cells (Isherwood, 2020). These findings further raise the significance of cortical plasticity as the cortex seems more adaptive to changes in light input and not anatomical areas that come first in relaying visual information.

On a cellular level, current research suggests that intrinsically photosensitive retinal ganglion cells (ipRGCs) mediate color processing (Raja, 2023), and can be considered a part of adaptive neuroplasticity in color blindness. While traditionally associated with non-image-forming functions such as circadian regulation, sleep, mood, and cognition, ipRGCs also receive input from cones and rods, and project to visual pathways, influencing both brightness and color percepts (Isherwood, 2020). This raises intriguing possibilities for their role in color vision deficiencies, particularly in dichromats lacking one cone type. Despite the absence of a full trichromatic signal, dichromats can still reliably categorize colors in ways that align with trichromats, a phenomenon attributed to sensory mechanisms and learned associations. Some studies suggest that dichromats can achieve a form of functional trichromacy over large visual fields by utilizing variations in spectral sensitivity across the retina or by incorporating rod-based signals (Isherwood, 2020). Although rods are generally considered "color blind," they have been shown to contribute to color perception under certain conditions. Given that ipRGCs integrate inputs from cones and rods, they may play an unrecognized role in color perception, particularly in individuals with color vision deficiencies who rely more heavily on alternative visual pathways (Isherwood, 2020). While the specific contributions of ipRGCs to color processing in dichromats remain unexplored, their distinct signaling properties may offer a valuable test case for investigating alternative mechanisms of color coding in the visual system.

Rehabilitation and Assistive Technologies

Given recent advancements in neuroscientific research on brain plasticity in colorblind individuals, this knowledge can be applied to developing rehabilitation and assistive technologies for optic injuries. Understanding neuroplasticity mechanisms in the visual cortex allows for creating personalized treatment approaches based on an individual's plasticity pattern. One common approach to aiding color blindness is using color-correcting glasses tailored to a person's specific receptor sensitivity. However, some researchers are also exploring gene therapy as a potential method to alleviate or even cure color blindness (Dougherty, 2024). More importantly, insights into brain plasticity extend beyond color blindness and can aid in treating brain injuries. Recent studies suggest that doctors can use neural training techniques, such as virtual reality therapy and constraint-induced movement therapy, to help the brain recover from damage (Zotey, 2023). A deeper understanding of neuroplasticity could make neural training a key component of non-invasive rehabilitation therapies. Additionally, research on the plasticity of the visual cortex may provide valuable insights into treating vision-related injuries (Barton, 2020) and conditions such as myopia (Tan, 2008).

Conclusion

In conclusion, color blindness offers a unique perspective on adaptive neuroplasticity, especially in the brain's visual cortex. Interestingly, the brains of individuals with different types of color blindness are more likely to experience structural and functional changes in the cortex and not in the retina. Hence, studying color blindness brings more attention to cortical neuroplasticity because these areas seem to play a more significant role in adaptation to vision impairment when compared to neurons and receptors involved in the first steps of color vision. Brain areas most involved in color vision adaptation are V1, V2, and V3 areas of the visual cortex, and research shows that V2 and V3 play a role in cortical reorganization of colorblind individuals. However, some studies also explore the extent to which cellular retinal structures like rods, ipRGCs, or neurons in LGN contribute to adaptive neuroplasticity as a response to altered light input. By using the knowledge about the adaptive neuroplasticity of the colorblind brain, scientists are looking to study neural training for rehabilitation and therapeutic technologies to treat brain injuries or visual impairments.

References

1. Ailioaie, Laura & Litscher, Gerhard. (2020). Molecular and Cellular Mechanisms of Arthritis in Children and Adults: New Perspectives on Applied Photobiomodulation. International journal of molecular sciences. 21. 10.3390/ijms21186565.

2. Andriani, R. (2024). Investigating neural processing of color in normal and impaired vision. medRxiv. https://doi.org/10.1101/2024.12.22.24319498

3. Arcaro, M J, and S Kastner. "Topographic organization of areas V3 and V4 and its relation to supra-areal organization of the primate visual system." Visual neuroscience vol. 32 (2015): E014. doi:10.1017/S0952523815000115

4. Barton, J. J. S., & Ranalli, P. J. (2020). Vision therapy: Ocular motor training in mild traumatic brain injury. Annals of Neurology, 88(3), 453–461. https://doi.org/10.1002/ana.25820

5. Dougherty, E. (2024). Color therapy. Harvard Medical School Magazine. Retrieved from https://magazine.hms.harvard.edu/articles/color-therapy

6. Fareed, M., Anwar, M. A., & Afzal, M. (2015). Prevalence and gene frequency of color vision impairments among children of six populations from North Indian region. Genes & Diseases, 2(2), 211–218. https://doi.org/10.1016/j.gendis.2015.02.006

7. Huff T, Mahabadi N, Tadi P. Neuroanatomy, Visual Cortex. [Updated 2023 Aug 14]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK482504/

8. Isherwood, Z. J., Joyce, D. S., Parthasarathy, M. K., & Webster, M. A. (2020). Plasticity in perception: Insights from color vision deficiencies. Faculty Reviews, 9, 8. https://doi.org/10.12703/b/9-8

9. Lee, B. B. (2008). The evolution of concepts of color vision. Neurociencias, 4(4), 209–224.

10. Neitz, J., Carroll, J., Yamauchi, Y., Neitz, M., & Williams, D. R. (2002). Color perception is mediated by a plastic neural mechanism that is adjustable in adults. Neuron, 35(4), 783–792. https://doi.org/10.1016/s0896-6273(02)00818-8

11. Pasmanter N, Munakomi S. Physiology, Color Perception. [Updated 2022 Sep 12]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK544355/

12. Puderbaugh M, Emmady PD. Neuroplasticity. [Updated 2023 May 1]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK557811/

13. Raja, S., Milosavljevic, N., Allen, A. E., & Cameron, M. A. (2023). Burning the candle at both ends: Intraretinal signaling of intrinsically photosensitive retinal ganglion cells. Frontiers in Cellular Neuroscience, 16. https://doi.org/10.3389/fncel.2022.1095787

14. Shichida, Yoshinori, and Take Matsuyama. "Evolution of opsins and phototransduction." Philosophical transactions of the Royal Society of London. Series B, Biological sciences vol. 364,1531 (2009): 2881-95. doi:10.1098/rstb.2009.0051

15. Simunovic, M. (2010). Colour vision deficiency. Eye, 24, 747–755. https://doi.org/10.1038/eye.2009.251

16. Tan, D. T., & Fong, A. (2008). Efficacy of neural vision therapy to enhance contrast sensitivity function and visual acuity in low myopia. Journal of Cataract and Refractive Surgery, 34(4), 570–577. https://doi.org/10.1016/j.jcrs.2007.11.052

17. Tregillus, K. E. M., Isherwood, Z. J., Vanston, J. E., Engel, S. A., MacLeod, D. I. A., Kuriki, I., & Webster, M. A. (2021). Color compensation in anomalous trichromats assessed with fMRI. Current Biology, 31(5), 936–942.e4. https://doi.org/10.1016/j.cub.2020.11.039

18. Zotey, V., Andhale, A., Shegekar, T., & Juganavar, A. (2023). Adaptive neuroplasticity in brain injury recovery: Strategies and insights. Cureus, 15(9), e45873. https://doi.org/10.7759/cureus.45873

About the Author

Yuliia Kohut is a Freshman in Bioengineering on a pre-medical track and a student from Ukraine. Apart from Brain Matters, on campus she is a Global Health executive member in the American Medical Student Association, and she is also a student volunteer at Carle Hospital. Yuliia is an undergraduate researcher in Dr. Best-Popescu lab at Beckman Institute, working on developing imaging tools for cellular neuroscience research. In her free time Yuliia enjoys cross-stitching, cooking Ukrainian food, and reading sci-fi novels. She joined the editing and writing team of Brain Matters to share her fascination with neuroscience with UIUC!

Interview with Two Professors

Written by Brianna Mae Huner

Introduction

While psychology and neuroscience have a long history, this knowledge is not set in stone, nor is it all-encompassing. New findings are always being presented, sometimes disproving generally accepted principles. The scientific method allows scientists to constantly be testing and retesting hypotheses, and if these hypotheses are wrong, then sometimes the solution is questioning the foundation on which these hypotheses are built upon. Asking questions is the entire foundation of science. The scientific model requires questioning what is known. The importance of asking these questions cannot be overstated, as investigation into these hypotheses can benefit the field and humanity at large. At the university level, professors have the opportunity to choose what they teach, including their own hypotheses. At the Psychology department at the University of Illinois, a variety of perspectives and hypotheses can be observed, coloring the general understanding of the topic of psychology and its concentrations, such as Neuroscience and Clinical Psychology.

Professor Justin Rhodes

Professor Justin Rhodes is a Professor at the Beckman Institute for Advanced Science and Technology and an Affiliate at the Carl R. Woese Institute for Genomic Biology. He earned his bachelor's degree in biology from Stanford and his PhD in zoology from the University of Wisconsin-Madison. Professor Rhodes has a research focus in cognitiveneuroscience and is researching how genes and the environment can affect behavior. His current lab research investigates how exercise can affect neurogenesis (the creation of new neurons) in the hippocampus. Rhodes frequently discusses these concepts in the classes he teaches, which are PSYC/NEUR 302 (Applied Neuroscience) and PSYC/NEUR/PHIL 433 (Evolutionary Neuroscience), which is also cross coded as IB 436.

Prof. Rhodes often collaborates with colleagues and wrote a chapter in the book of his doctoral advisor, Dr. Theodore Garland Jr., on behavior and neurobiology. Rhodes described the importance of splitting duties based on expertise when collaborating on academic writing, writing sections of papers dedicated to concepts he has the most experience in, and leaving space for collaborators with more experience in other topics. Recently, he wrote a paper with Dr. Ki Yun Lee and Dr. Taher Saif about the involvement of astrocytes in the muscle fiber contraction-hippocampal development network, which showed that astrocytes may mediate this relationship. Prof. Rhodes expressed his interest in the unknown roles of glia in the brain, highlighting how microglia are seen to have specific responses to different levels of an organism engaging in physical activity. Cumulatively, the function of microglia is altered by the effect of an organism running. He would like to see an investigation into the role of signaling in the blood in this effect. Rhodes has conducted an abundance of research on the hippocampus and has some of his own propositions to its functions.

During intense physical activity, it has been observed that the hippocampus, typically associated with learning and memory, becomes activated. The level of activation in the hippocampus appears to have a positive relationship with the level of intensity of the activity, further indicating that these two are related. This phenomenon is a subject of curiosity and debate among neuroscientists, as the current understanding of the hippocampus and intense physical activity seem to be independent processes. There is evidence to show that physical activity could encourage neurogenesis in the hippocampus. But this evidence is not sufficient to explain how the level of intensity seems to correlate with the level of activation in the hippocampus.

The classic interpretation behind this activation is that the hippocampus is acting as a part of the sensory system. This hypothesis assumes that the hippocampus activates in response to speed, because if the animal is moving through space faster, they will need to sample their environment faster to keep their spatial map up to date. The spatial map is the brain's representation of the spatial environment surrounding it, which is used to support functioning in the environment and relating to the space one is in. The mechanism used to explain this activation is that during hippocampal activation, the hippocampus creates waves of activity, with the most salient, or powerful and effective, learning occurring at the peaks and troughs of these waves. This supposes that to increase the rate of learning, the hippocampus would need to increase the frequency of these waves to maximize learning potential.

Prof. Rhodes believes that the hippocampus is serving a different purpose in this intense activity system, acting as a motivator of this system, instead of serving as a reactionary sensory organ. He hypothesizes that the hippocampus acts as an intensity generator, allowing the animal to move at intense speeds by motivating the body to move. It should be noted that this form of motivation is not of the higher order of consciousness, where people choose to move intensely because of their understanding of the health benefits of such activity or because they want to escape something. Instead, Prof. Rhodes and his colleagues believe that the hippocampus is the organ needed to allow the body to move at such intense speeds when necessary. There have been several studies conducted testing this hypothesis, and a wealth of evidence collected to support it.

Recordings taken in the entorhinal cortex reveal that hippocampal activity precedes intense movement. This evidence could not be explained by the popular hypothesis of the hippocampus acting as a reaction to the activity, as this movement could not cause neural activity that precedes it.

The sensory hypothesis also does not explain why this hippocampal activation does not occur when a subject is being moved through an environment at high speeds whenthey are not running. In a study where test mice were placed in transportation carts (analogous to a car for a human) and moved at a rapid rate, the level of activation seen in intense activity is not observed in the hippocampus, despite the need to rapidly update the spatial map still being present. However, this is a potential side effect of human evolution adjusting far slower than human technological development. This lack of activation when moving quickly through technological means could be explained by human evolution not progressing past the stage where the only way to get around quickly was through intense activity such as running. These recordings of neural activity support the hypothesis that Prof. Rhodes believes. Stimulating these areas, instead of simply observing them, could also generate evidence to support this theory.

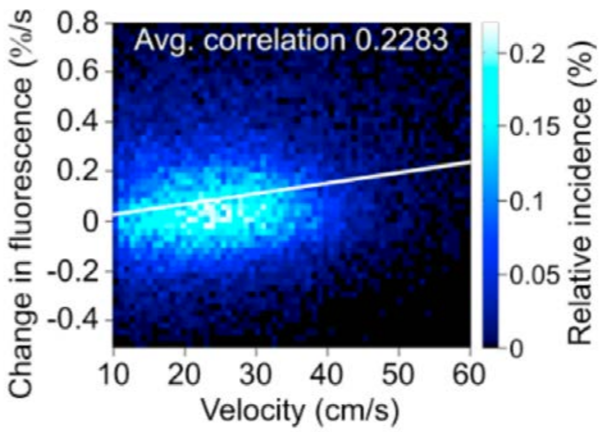


Figure 1. Positive correlation of GCaMP5 fluorescence slope and velocity.

Prof. Rhodes cited a study conducted with Dr. Stefan Remy, a professor at the University of Bonn in Germany. This study was conducted in mice using optogenetics, a revolutionary new technique that allows for the stimulation of specific cells using light. The study used optogenetics to stimulate the hippocampus of mice while on a treadmill. When the hippocampus was stimulated, the mice began to move around and run at an intensity that matched the level of activation in the hippocampus. This evidence supports the idea that the hippocampus supports motivation and ability to move quickly.

This theory has its detractors, however, who call on the current understanding of the motor system and clinical history. The hippocampus is not traditionally connected to the movement system in the field of neuroscience. Patient H.M., a famous patient in the history of psychology, was known for having large portions of his temporal lobe removed from his brain, including his hippocampus. While Patient H.M. is widely known to have lost many of the abilities traditionally associated with the hippocampus, such as the ability to form new memories, he was able to move quickly or engage in intense movement after his operation.

Though, this possibility was not officially studied with HM. The hypothesis believed by Prof. Rhodes of hippocampal involvement provides evidence for why this may have been. This data has also been seen in studies, as the loss of the ability for intense movement when the hippocampus is lesioned has also been observed in experiments where rats have their hippocampus suppressed via anesthetics. The combination of evidence discussed, through studies of recording and stimulation in the hippocampus as well as lesion studies, supports Rhodes's motivator hypothesis. If hippocampal activation precedes intense movement, then it cannot be caused by intense movement, nor can it explain why similar hippocampal activation in response to non-neural-motor forms of rapid movement is not seen. In addition, his idea supports why directly stimulating the hippocampus induces intense motor activity. Finally, while removal of the hippocampus does not have all-encompassing effects on the motor system, it seems to remove the organism's ability to engage in intense movement. The commonly held sensory hypothesis of the relationship between hippocampal activation and intense physical activity does not explain this evidence. Discussions such as these encourage neuroscientists to look deeper into the validity of these hypotheses.

Prof. Rhodes's research and hypotheses of the hippocampus can aid in growing the scientific understanding of the brain region associated with some of the most damaging effects of aging. The hippocampus is the first part of the brain to begin deteriorating as humans age, losing about 1% of it every year after the age of 20. The hippocampus is the brain region most associated with dementia, but it is also one of only two brain regions that is able to create new neurons throughout the human lifetime. Studies have shown that hippocampal exercise may be beneficial for retaining hippocampal mass as humans age as regular strenuous exercise can promote neurogenesis in the hippocampus. Evaluating the current knowledge of the hippocampus, especially in relation to exercise, could grant a revolutionary tool in the treatment of neurodegenerative diseases, as well as the regular struggles of aging and memory impairment.

Professor Thomas Kwapli

Professor Thomas Kwapil is the Director of Clinical Training and an Associate Head of the Psychology Department at the University of Illinois. He describes his role as being responsible for ensuring that the Clinical-Community program runs smoothly, as well as acting as a liaison with the department head. The Clinical-Community program comes with extra responsibilities, as it requires accreditation, which requires reporting to outside agencies. He received his bachelor's degree in psychology from Louisiana State University, and his M.S. in psychology and PhD in clinical psychology from the University of Wisconsin-Madison. Prof. Kwapil is the current supervising professor for PSYC 238 (Psychopathology and Problems in Living) and PSYC 239 (Community Psych). This is a role that rotates among different professors in the department. Instead of being the instructor for the course, the supervising professor supports and supervises the graduate students who teach the course. Professor Kwapil also regularly teaches a PSYC 496 (Adv Current Topics in Psych) course on schizophrenia-spectrum disorders, which is his area of interest.

Schizophrenia-spectrum disorders include schizophrenia and other psychotic disorders, including schizophreniform disorder and brief psychotic disorder, as well as schizotypal personality disorder. It is seen through abnormalities in "positive" symptoms (including delusions and hallucinations), disorganized symptoms (such as disorganized thinking, speech, and behavior), and "negative" symptoms (such as avolition and diminished emotional expression). An estimated 3-4% of the population suffers from schizophrenia spectrum disorders. Many psychologists, especially those whose perceptions are colored by the categorical nature of the Diagnostic and Statistical Manual of Mental Disorders (DSM), would see these as categorical disorders, following a yes/no binary. Prof. Kwapil disagrees with this notion.

Figure 2. SEQ Figure * ARABIC 2. Schizophrenia Spectrum Disorders. Giovanni, V. (2015).

It is widely believed that other disorders, such as depression and anxiety, exist on a spectrum of intensity and subclinical presentations, yet this line of thinking is not often extended to schizophrenia-spectrum disorders. Many people report experiencing anxiety or depression. With some, a more clinical expression is seen. With others, these symptoms are less intense, do not last as long, and/or cause less impairment. All are experiencing these symptoms; some are simply experiencing them on a subclinical level. The same can be true for schizophrenia-spectrum psychopathology. Even when it comes to clinical schizophrenia-spectrum disorder presentations, categorical measures fall short to dimensional measures. Those with schizophrenia may experience psychotic episodes, which remit and return, similar to depressive episodes seen in Major Depressive Disorder. An all-or-none categorical form of thinking, in which a subject is either in or out of an episode, may be easier to conceptualize, but is not entirely accurate to describe this phenomenon. Often when people remit from episodes, their expression of symptoms doesn't go from disordered to regularly functioning, but rather a less intense symptomology or degree of impairment. Where the categorical model fails to encapsulate these observations, Prof. Kwapil has a more fitting explanation.

Prof. Kwapil and his colleagues support a dimensional model, in which schizophrenia-spectrum disorders and subclinical presentations of schizophrenia exist on a continuum, called schizotypy. Prof. Kwapil and his colleagues are pursuing an effort to reconceptualize schizophrenia and related disorders as not simply extreme and rare disorders, but also milder presentations. Often conceptualized as extreme manifestations in clinical cases, milder forms of positive, disorganized, and negative symptoms can also be seen. These subclinical symptoms can develop and change in a variety of ways, if at all. Some of these milder symptoms go on to develop into a clinical, disordered presentation. Some continue to have these symptoms, but they do not get worse or interfere with functioning. Others will have these symptoms come and go, sometimes never appearing again. A key difference between milder symptoms and clinical symptoms when it comes to, for example, delusional thinking, is in conviction. Someone with a clinical expression of delusional thinking may be convinced that someone is stealing their thoughts or threatening them, whereas someone with a subclinical expression of delusional thinking may wonder or suspect if it may be possible but are unsure.

Prof. Kwapil's method for studying the dimensional model involves a complex process that he has implemented into his research. First, development of a conceptual model for understanding the dimensions of schizotypy is needed. Models have been developed to measure the dimensions of positive, disorganized, and negative schizotypy in order to identify people who exist on this scale and how their symptoms should present. When observed in subclinical expressions, which do not cause impairment, those being studied are not seen as patients, but rather personality traits. People can be elevated on one, two, or all three dimensions. For example, someone who may be high in the positive dimension, experiencing magical beliefs or strange perceptual experiences, may be low in negative and disorganized dimensions, such that they would not be experiencing the symptoms such as flattened affect, anhedonia, or difficulty organizing and executing thoughts and emotions. Participants for a study of subclinical presentations of schizotypy are typically found through extensive interview studies or questionnaire studies focusing on schizotypal traits, personality, and emotion. One of the testing methods favored by Prof. Kwapil is Experience Sampling Methodology, or ESM. ESM is a measurement that uses a system of daily surveys sent out multiple times each day that assesses the dynamic system of emotional and psychological phenomena throughout the day. This is a more effective measurement system for seeing a larger picture of a participant's life by measuring them over a larger period of time than a single lab interview session, making it a fitting choice for studying subclinical presentations of psychological disorders.

Schizotypy, like many dimensional models of clinical disorders, offers a useful conceptual model of schizophrenia spectrum disorders that has advantages over traditional categorical models. Human behavior and emotions have a complexity and nuance that cannot be entirely captured through a yes/no binary. Similar to anxiety and depression, which are generally accepted to be experienced both clinically and subclinically, symptoms of schizophrenia spectrum disorder can be observed on a lower impairment level among those who do not fit the criteria for schizophrenia spectrum disorders. Prof. Kwapil has been studying schizotypy for years and has developed an effective model of study of the dimensions of schizotypy. With models such as these, the scientific community can move on to a better understanding of the truly dimensional model of psychological disorders and their subclinical presentations.

Prof. Kwapil's proposed model of schizotypy can advance the understanding of schizotypy, allowing for an improved early detection system and potentially negating some of the stigma associated with these disorders. An estimated 10% of people experience subclinical expressions of schizotypy, which is a large portion of the population and merits investigating the presentation and mechanism behind these symptoms. Identifying these individuals could be instrumental in instituting early intervention services for those who will eventually develop a clinical presentation of schizotypy. However, those with schizophrenia spectrum disorders have a heavy stigma against them among the general public, and it is important to acknowledge this during research. When trying to identify potential prodromal symptoms, which can help with preventing these symptoms from developing to a clinical level, those identified with subclinical symptoms could put at risk of losing social support systems, losing their jobs, and potentially being prevented from being covered by insurance because of the association with these disorders. Being identified as being at risk of developing heavily stigmatized disorders such as schizophrenia or its related disorders carries a much difference social weight than those identified as being at risk for the development of less stigmatized illnesses, such as breast cancer. As those with schizophrenia are often mischaracterized as dangerous or violent. However, as the understanding of schizotypy grows through research, it is possible for education about schizotypy to be spread outside of the scientific community, placing logic and understanding in place of fear.

About the Author

Brianna Mae is a Junior at the University of Illinois majoring in Clinical/Community Psychology. She became involved in Brain Matters to gain more experience researching and writing about the current research in Neuroscience. When she is not writing for Brain Matters, she is also involved in Dr. Kwapil's Project on Life Experiences Lab, and is the Treasurer for the Psychology Research and Community Club (PRACC). Brianna Mae is hoping to pursue a PhD in Clinical Neuropsychology and conduct research about the neurological basis behind different clinical disorders.

Amusia: The Science Behind Tone Deafness

Written by Emily Aldrich

What is Tone Deafness?

For most, the ability to distinguish basic pitch, rhythm, and melody of music is something that comes naturally. The brain is attuned to hearing certain sounds and perceiving them as music, allowing for listeners to enjoy what they are listening to as well as imitate what they hear. This seemingly simple, unconscious knowledge of music for individuals is something that about 2.5% of the population lacks (Lehmann et al., 2015). Congenital amusia, often referred to as “tone deafness”, is a disorder present at birth that is characterized by the impairment of musical perception. Although amusic individuals know that the sound they are hearing is supposed to be a song, the concept of music as a whole is lost on them. Those with congenital amusia have an inability to perceive music as a coherent network of melodic elements (Szyfter & Wigowska, 2021). Instead, they recognize it only as disorganized noise in the environment, and can have deficits in perception of tune, melody, and rhythm – all factors that make up music.

Audition Process

Auditory processing occurs during any moment where sound is made and can be perceived, triggering the activation of neural pathways in the brain. Sounds start as air pressure waves that go through the ear canal, which is then detected by hair-like projections called stereocilia, located on the basilar membrane of the cochlea (Kulsoom & Karim, 2021). At this point in processing, these vibrations are transformed into electrical signals that are then sent to neurons in the spiral ganglion, whose axons form the auditory nerve (Kulsoom & Karim, 2021). The auditory nerve then sends the signal to the brainstem’s auditory nuclei, a relay station for these signals to then be sent out to numerous different areas of the brain for further processing. Although these signals extend far out into different parts of the brain, this auditory process occurs all in the span of milliseconds.

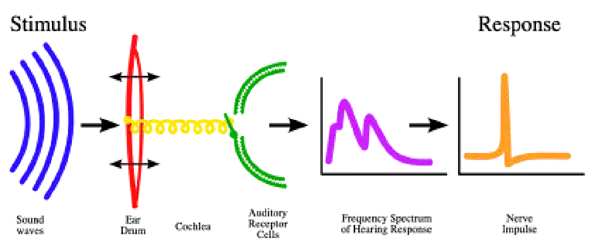


Figure 1. Audition process where sound waves become nerve impulses (Gollisch & Herz, 2005).

Physiological Aspects

Biological differences between those with amusia and the general population have been studied to better understand how tone deafness manifests in both the nervous system and the genetics of the affected group. One area that has been shown to have ties with amusia and general musical ability is the brainstem (Lehmann et al., 2015), a structure that sits just below the brain and connects to the spinal cord. The brainstem mediates many pathways of neuronal activity, allowing for the coordination of different brain functions. Researchers used complex auditory brain responses (cABRs) to measure the subcortical processing of complex sounds like tones, speech, and in this case, music (Anderson & Kraus, 2013). For amusic individuals who process music irregularly, their cABRs have shown decreased spectral amplitude meaning frequencies were weaker in magnitude, and slower onset responses; the stronger the disability in musical processing, the stronger these differences show up as cABRs (Lehmann et al., 2015). These areas of deviation from the norm show that there are specific locations and pathways where music is processed. When these pathways are not processed correctly, this may elicit a misunderstanding of sound in a way that amusics perceive as disorganized and not melodious. This information indicates that the physiological differences between amusics and non-amusics affect how their perceptions of complex sounds like music.

Genetic Components

There is likely a genetic component regarding congenital amusia, as many people who have this disorder are shown to have other family members with similar musical deficits. To learn more about the genetic underpinnings of congenital amusia, researchers began to study self-identified amusic individuals who also did poorly on the Montreal Battery of Evaluation of Amusia (MBEA), a test to determine the level of musical capability one has by assessing scale, contour, interval, rhythm, metric, and music memory–all components of music processing (Nunes-Silva & Haase, 2012). They had both amusic and non-amusic control groups encourage family members to take an assessment similar to MBEA that tested the same musical skills to determine whether or not these family members would have a comparable score to their relative. This study deduced that 39% of first degree relatives of people with amusia also have the same disorder, while only 3% of relatives have amusia in the control group (Peretz et al., 2007). The heritability of amusia is very evident, meaning genetics plays an important role in the phenotype of congenital amusia.

Sociocultural Impacts of Amusia

A highlighting feature of amusia is the consistent inability to recognize musical tunes, which also extends to music memory. It is strongly connected to deficits in recognizing small pitch changes. A noteworthy feature from amusia studies is that although difficulties in musical perception does not extend to the language realm for non-tonal languages like English, amusical people who use tonal languages like Mandarin or Vietnamese do slightly struggle in speech perception. In tonal languages, the pitch or tone of a syllable itself can change the meaning of words. In Mandarin, for example, the syllable /ma/ can mean either "mother", "horse", "hemp", or "to scold" depending on which tone is used, as shown in Figure 2 (Li et al., 2021). Amusics who do speak tonal languages are able to produce the correct tones when creating speech, but have greater difficulty in accurately imitating pitches for tones they hear (Liu et al., 2013). Although they are still able to produce language at a proficient level, there is still some overlap between the presence of amusia and speech imitation in tonal languages because they are so reliant on the ability to use the correct pitches and match them accordingly.

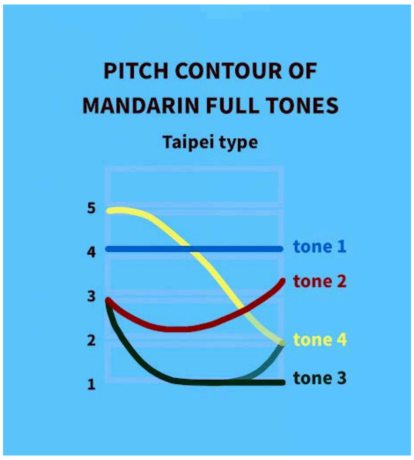


Figure 2. Standard Chinese tone contours (Wikimedia Foundation, 2021).

Further Look into Amusia

The progression of our understanding of congenital amusia has gone very far in just the past few decades. There have been many groundbreaking studies that further explain both the origins and long-term effects of this cognitive deficit. It is essential that we learn more about this lifelong disorder. From the progression in research that we have seen so much of, there are even more thoughts and questions that are important to answer. We now know there is a strong genetic component of this disorder. Future research can help identify amusia-related genes in individuals to learn more about how it is inherited, as well as finding treatments to target amusia. Additionally, it would be interesting to dig deeper in the social impact amusia has on its individual–particularly how the emotions that are elicited when listening to a certain song compares to controls, as well as its possible impact on individuals who speak pitch-accent languages. Through further research, we could potentially gain greater understanding of how amusics perceive and experience the world around them.

References

1. Anderson S, Kraus N. The Potential Role of the cABR in Assessment and Management of Hearing Impairment. Int J Otolaryngol. 2013;2013:604729. doi: 10.1155/2013/604729. Epub 2013 Jan 30. PMID: 23431313; PMCID: PMC3572655.

2. Gollisch, T., & Herz, A. (2005, January). *File:processing-of-sound.svg*. Wikimedia Commons. https://commons.wikimedia.org/wiki/File:Processing-of-sound.svg

3. Kawamura M, Miller MW. History of Amusia. Front Neurol Neurosci. 2019;44:83-88. doi: 10.1159/000494955. Epub 2019 Apr 30. PMID: 31220839.
4. Kulsoom B, Karim N. Sound: Production, perception, hearing loss and treatment options. J Pak Med Assoc. 2022 Apr;72(4):725-732. doi: 10.47391/JPMA.4051. PMID: 35614609.
5. Lehmann A, Skoe E, Moreau P, Peretz I, Kraus N. Impairments in musical abilities reflected in the auditory brainstem: evidence from congenital amusia. Eur J Neurosci. 2015 Jul;42(1):1644-50. doi: 10.1111/ejn.12931. Epub 2015 Jun 18. PMID: 25900043.
6. Li Y, Tang C, Lu J, Wu J, Chang EF. Human cortical encoding of pitch in tonal and non-tonal languages. Nat Commun. 2021 Feb 19;12(1):1161. doi: 10.1038/s41467-021-21430-x. PMID: 33608548; PMCID: PMC7896081.
7. Liu F, Jiang C, Pfordresher PQ, Mantell JT, Xu Y, Yang Y, Stewart L. Individuals with congenital amusia imitate pitches more accurately in singing than in speaking: implications for music and language processing. Atten Percept Psychophys. 2013 Nov;75(8):1783-98. doi: 10.3758/s13414-013-0506-1. PMID: 23877539.\
8. Nunes-Silva M, Haase VG. Montreal Battery of Evaluation of Amusia: Validity evidence and norms for adolescents in Belo Horizonte, Minas Gerais, Brazil. Dement Neuropsychol. 2012 Oct-Dec;6(4):244-252. doi: 10.1590/S1980-57642012DN06040008. PMID: 29213804; PMCID: PMC5619336.
9. Peretz I, Cummings S, Dubé MP. The genetics of congenital amusia (tone deafness): a family-aggregation study. Am J Hum Genet. 2007 Sep;81(3):582-8. doi: 10.1086/521337. Epub 2007 Jul 20. PMID: 17701903; PMCID: PMC1950825.
10. Szyfter K, Wigowska-Sowińska J. Congenital amusia-pathology of musical disorder. J Appl Genet. 2022 Feb;63(1):127-131. doi: 10.1007/s13353-021-00662-z. Epub 2021 Sep 21. PMID: 34545551; PMCID: PMC8755656.
11. Vuust P, Heggli OA, Friston KJ, Kringelbach ML. Music in the brain. Nat Rev Neurosci. 2022 May;23(5):287-305. doi: 10.1038/s41583-022-00578-5. Epub 2022 Mar 29. PMID: 35352057.
12. Wikimedia Foundation. (2021, December). File:Mandarin Tone Contours (beijing standard).svg. Wikipedia. https://en.m.wikipedia.org/wiki/File:Mandarin_tone_contours_(Beijing_standard).svg

About the Author

Emily Aldrich is a Freshman majoring in Neuroscience with minors in Linguistics and Psychology on the pre-med track. Emily joined Brain Matters to gain a deeper understanding of the brain through exploring current research topics in neuroscience. In her free time, she enjoys listening to music, reading, and spending time with friends.

The Difference Between Children's Learning Abilities With and Without ADHD

Written by Leah Rupp

Introduction

Attention-deficit hyperactivity disorder (ADHD) is one of the most common neurodevelopmental disorders found in children (Cleveland Clinic, 2023). It is a developmental disorder that can interfere with social and school-related activities and can be diagnosed as young as 2 years of age. Low amounts of gray matter and neurotransmitter γ-aminobutyric acid (GABA) have all been connected to ADHD symptoms. Gray matter, primarily composed of neuronal cell bodies, dendrites, and unmyelinated axons, is commonly found in lower amounts in patients with ADHD. Another cause of ADHD is lower concentrations of GABA.

Physicians most commonly screen for ADHD by using the Diagnostic and Statistical Manual, Fifth edition (DSM-5), a test consisting of a series of yes or no questions. Individuals aged 16 or younger who answer 'yes' to six or more of the questions meet the diagnostic criteria for ADHD. These questions are designed to assess common behaviors associated with ADHD, such as, “often does not seem to listen when spoken to directly,” “often has trouble holding attention to tasks or play activities,” and “often fidgets with or taps hands or feet” (Cleveland Clinic, 2023). It can be difficult to diagnose ADHD because many symptoms are very similar to various anxiety and mood disorders. Moreover, ADHD affects young children mostly in school (Cleveland Clinic, 2023), where challenges sitting still for extended periods can impact their learning experience.

For children six years and younger it is recommended to try behavior therapy rather than start ADHD drugs (Cleveland Clinic, 2023). Parents can try to better manage children's behavior by creating a routine, utilizing prizes when goals are achieved, and limiting choices. However, for children older than six, medical treatments are recommended (Cleveland Clinic, 2023). ADHD medications consist of stimulants and non-stimulants, with non-stimulants being less effective. Common stimulants are methylphenidate and amphetamine, which create a calming effect on children with ADHD. Individuals may only see results after weeks of taking the drug (Cleveland Clinic, 2023).

Gray’s Matters Effect on ADHD

Dr. Luke Norman, a scientist at the National Institute of Mental Health (NIMH), discovered that ADHD is linked to irregular connections between the brain’s frontal cortex and information processing centers. The cerebral cortex, the outermost layer of the brain, plays a critical role in processing information, supported by the dendrites that play a key role in receiving chemical messages from other nerve cells. According to the Cleveland Clinic, this region of the brain is also responsible for “memory, learning, and decision-making” (Cleveland Clinic, 2024). Individuals with ADHD have a smaller amount of gray matter in addition to a decreased volume of specific cortical regions, as shown in Figure 1. This may help explain why individuals diagnosed with ADHD often exhibit a shorter attention span and may struggle to maintain focus for extended periods because of the lower amounts of dendrites that receive chemical messages.

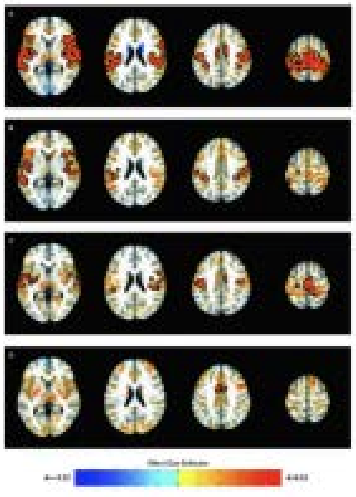


Figure 1. Smaller amounts of gray matter seen in individuals with ADHD, images C and D vs those without ADHD, images A and B

The frontal cortex of individuals with ADHD tends to develop at a much slower rate than individuals with a neurotypical brain. Children with neurotypical brains may have higher rates of neuroplasticity, which can increase their ability to retain information efficiently (Puderbaugh, 2023). According to the National Institute of Health, Neuroplasticity is "a process that involves adaptive structural and functional changes to the brain" (Puderbaugh, 2023). Children are often able to learn more quickly than adults due to greater neuroplasticity; however, this ability diminishes with age. During the early years of life, a child's brain is rapidly absorbing and processing new information like a sponge, leading to learning a language more readily than an individual in their 80s for example.

Studies on γ-aminobutyric acid

Dr. Sebastian M. Frank, a researcher at Brown University, discovered that the increased amount of neurotransmitter γ-aminobutyric acid (GABA) in children helps them retain new information that is learned. According to the Cleveland Clinic, GABA is an inhibiting neurotransmitter that "creates a calming effect" as it stops chemical messages from being sent from one nerve cell to another.

Dr. Richard A. Edden, a neuroradiologist at The John Hopkins University School of Medicine, conducted a study connecting GABA amounts and those diagnosed with ADHD. The goal of the study was to connect GABA concentration in an individual and whether or not they have ADHD. Patient's brains were scanned with a 3-Tesla MRI scanner. Children from ages 8-12 with ADHD were compared with children with neurotypical brains. The findings, shown in Figure 2, reveal a notable difference in GABA concentrations between children with and without ADHD. Children without ADHD have higher levels of GABA, which may contribute to their ability to learn efficiently and maintain focus. In contrast, children with ADHD tend to have lower GABA concentrations, potentially reducing the 'calming effect' that GABA provides, as described by the Cleveland Clinic. This difference can negatively impact their ability to stay focused in school, making it challenging to engage in prolonged learning activities. With the correlation between GABA and ADHD noticed, potential GABAergic therapies could decrease symptoms of ADHD seen in kids (Edden, 2012).

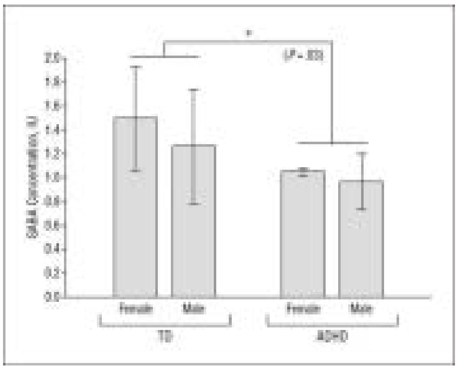


Figure 2. The bar graph displays a statistically significant difference in GABA concentration between the Female and Male TD group (neurotypical) as compared to the Female and Male ADHD group.

Conclusion

By studying gray matter and GABA, ADHD medication can better target what is exactly causing ADHD. Qelbree and Kapvay are newer drugs that target grey matter to help lower hyperactivity as seen in those diagnosed with ADHD. However, so much still needs to be discovered on what is the most efficient way to reduce ADHD symptoms, as many of these common drugs have drastic side effects such as nausea, headaches, and tiredness. According to the CDC, six million children between the ages of 2-17 in America have been diagnosed with ADHD. The implications of this diagnosis and its treatment negatively affect them academically and socially. From the connections found between grey matter and GABA in ADHD, more drugs could better target these areas to treat ADHD.

References

1. Norman, L. (2024, March 13). *NIH researchers identify brain connections associated with ADHD in Youth*. National Institutes of Health.
https://www.nih.gov/news-events/news-releases/nih-researchers-identify-brain-connections-associated-adhd-youth (Figure 1)

2. Edden, R. A. E., Crocetti, D., Zhu, H., Gilbert, D. L., & Mostofsky, S. H. (2012, July). Reduced gaba concentration in attention-deficit/hyperactivity disorder. Archives of general psychiatry. https://pmc.ncbi.nlm.nih.gov/articles/PMC3970207/ (Figure 2)
3. Centers for Disease Control and Prevention. (n.d.). Treatment of ADHD. Centers for Disease Control and Prevention. https://www.cdc.gov/adhd/treatment/index.html
4. ADHD vs neurotypical brains: Mental health academy. ADHD vs Neurotypical Brains | Mental Health Academy. (n.d.). https://www.mentalhealthacademy.com.au/blog/adhd-vs-neurotypical-brains-implications -for-therapists
5. Puderbaugh, M. (2023, May 1). Neuroplasticity. StatPearls [Internet]. https://www.ncbi.nlm.nih.gov/books/NBK557811/
6. Professional, C. C. medical. (2024, May 1). Gamma-aminobutyric acid (GABA): What it is, Function & Benefits. Cleveland Clinic. https://my.clevelandclinic.org/health/articles/22857-gamma-aminobutyric-acid-gaba
7. Commissioner, O. of the. (n.d.). The FDA has approved medications to help reduce the symptoms of ADHD. U.S. Food and Drug Administration. https://www.fda.gov/consumers/consumer-updates/treating-and-dealing-adhd
8. Why do children learn more quickly than adults? new study offers clues. Brown University. (2024, November 13). https://www.brown.edu/news/2022-11-15/children-learning
9. professional, C. C. medical. (2024c, September 1). Cerebral cortex: What it is, Function & Location. Cleveland Clinic. https://my.clevelandclinic.org/health/articles/23073-cerebral-cortex
10. Attention-deficit/hyperactivity disorder (ADHD). Cleveland Clinic. (2023, February 22). https://my.clevelandclinic.org/health/diseases/4784-attention-deficithyperactivity-disorde r-adhd

About the Author

Leah Rupp is a freshman at the University of Illinois in Urbana-Champaign studying Molecular and Cellular Biology within the honors concentration. Leah joined Brain Matters to get the opportunity to learn and write about new neuroscience research. Leah is also a Stress Management Peer with McKinley Health Center and a volunteer with the Food Assistance and Well-being Program. In her free time, Leah enjoys running and playing the piano. Her career aspiration is to become a physician.

How Do We Measure Intelligence?

Written by Ananya Sampathkumar

Introduction

Intelligence is something that scientists have studied for centuries. From a neuropsychological standpoint, intelligence can be defined as one's abilities to adapt and change according to different environments and learn from one's experiences (Sternberg, 2012). This somewhat vague definition has led to multiple ways to measure it over the years, with each one prioritizing and measuring different aspects of cognitive function.

Alfred Binet was the father of the IQ test, working with Theodore Simon to create the Binet-Simon IQ test in 1905. This test consisted of 30 questions and had many issues as it did not consider the complexities of IQ and intelligence and therefore, could not provide a holistic result (Sternberg & Jarvin, 2015). However, this IQ test stands as the first prolific intelligence measurement exam. Consequently, it has acted as a basis for almost every IQ exam that has followed.

Some examples of IQ tests that have been derived from the Binet-Simon test are the Stanford-Binet test in 1916 (Sternberg & Jarvin, 2015) and the Army Alpha/Army Beta exams used by military generals during World War I (Warne et al. 2019). The Stanford-Binet IQ test would provide a single number, an IQ quotient, that represented an individual's place on the scale. The Army Alpha test was written, while the Beta version was pictures, for soldiers who could not read. They were used in determining what soldiers were suited for—which positions and leadership roles. However, the most prominent and used example is the Wechsler Intelligence Scale (WAIS), developed by David Wechsler (Niileksela & Reynolds, 2019), and has many uses.

IQ: An Introduction

Due to the complexity of intelligence, research on the neurobiological aspect of intelligence is reliant on two main forms of information gathering: brain imaging and genetic studies. These studies are thought to be able to help researchers learn more about higher-level cognitive processing. Several brain scans have shown that IQ scores were correlated with intracranial, cerebral, temporal lobe, hippocampal, and cerebellar volumes, which essentially makes up the entirety of the brain (Goriounova & Mansvelder, 2019). However, Voxel-based morphometry (VBM), a neuroimaging technique that allows scientists to make estimations about the spacing and distribution of differences between important or central brain regions, has been used to study IQ in the brain as well. Goriounova and Mansvelder (2019) found positive correlations between intelligence (found using the WAIS) and cortical thickness were seen in several different regions of the temporal and frontal lobes.

Studies like these show the many different parts of the brain that are affected by and interact with one another. As a result of the complex interplay between a multitude of brain regions, it is essential to utilize a complex and comprehensive IQ examination in order to best measure the trait of intelligence (Colom et al. 2010). These intricacies need to be reflected in the IQ and intelligence examinations that psychologists and neuroscientists use to study higher-order cognition.

The current most popular IQ test in usage is the WAIS-V, which David Wechsler developed to study the cognitive abilities of adults (Sternberg & Jarvin, 2015). This test provides a comprehensive measure of one's cognitive functioning in both verbal and performance situations. This scale assesses several unique types of intelligence through five main areas: Fluid Reasoning, Processing Speed, Verbal Comprehension, Visual-Spatial Ability, and Working Memory. Each of these sections contains several different tests within them. Along with these, two broader areas are scored: Full-scale IQ, which is based on the total combined performance of the past five categories and General Ability Index (GAI), which is only based on the Perceptual Reasoning Index and Verbal Comprehension Index. Together, all of this is expected to provide a holistic view of one's IQ (Niileksela & Reynolds, 2019).

These scores are calculated through a complex process. On the WAIS, the scores of the test-taker are compared to the scores of others within their general age group. From there, the average score is set around 100, and having a score of 90 to 110 is considered average intelligence (Loring & Bauer, 2010).

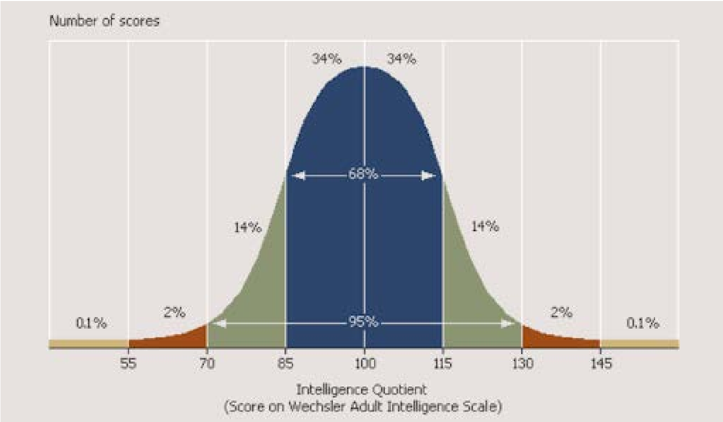


Figure 1. (SFU, 2005)

Benefits and Downsides

The WAIS is incredibly useful for determining one's intellectual abilities, identifying learning disabilities, and has been utilized to guide education and learning throughout the many years of its use (Koriakin et al. 2014). This test can also be used to diagnose and assess traumatic brain injuries, and is used in clinical trials and studies to learn about the effects of TBIs (Lida et al, 2021).

> **"... useful for determining one's intellectual abilities, identifying learning disabilities, and has been utilized to guide education and learning..."**

In addition, the WAIS has good test-retest reliability (Watkins & Smith, 2013). This ensures that the results that scientists are getting through these tests are accurate and consistent between each person. This consistency is incredibly important, especially when it comes to situations where an individual's data will be compared against that of the "entire population."

On the other hand, the WAIS does not cover every form of intelligence believed to exist. For example, creativity is not tested on the exam (Niileksela & Reynolds, 2019) but is considered to be a crucial type of intelligence (Dechaume et al. 2024). This exclusion of important intelligence factors may lead to several people high in related types of intelligence doing poorly on the WAIS.

Additionally, there are many concerns that factors such as race, gender, or nationality could play a role in testing and lead to unfair results. These exams are administered by psychologists, and their inherent biases could lead to results being different than the expected or accurate results from the person. This is seen most prominently regarding the Black and Hispanic American communities, which often face external stereotyping and discrimination. This bias can be seen through the Cultural Test Bias Hypothesis (CTBH), the idea that any gender, ethnic, or other minority groups that perform differently on the mental examinations are due to flawed methodology that was used to make and administer the exam (Reynolds et al, 2021).

Finally, the reliability of IQ exams has been brought into question. While all IQ exams have the same structure and format for the questions and sections, there are many external factors that could play a role in test results. One pilot study of the WAIS-II, one of the older versions, showed that testing reliability changed depending on the testing conditions. Elements such as which psychologist is proctoring the exam or what room the examination is being taken in can greatly affect one's score and results (Worhach et al., 2021).

Conclusion

Intelligence has been studied by scientists for centuries, and the complex interplay between brain regions is still not understood today. Despite testing flaws, scientists continue to use IQ testing to learn more about intelligence levels because they are a useful tool to reflect what is understood about the trait of higher-order cognitive functioning. In the future, scientists will use tools such as functional brain imaging in order to learn more about intelligence's changes to the brain. For example, a recent study that used lesion-mapping technology and WAIS-III index scores showed scientists that higher scores were correlated with more localized regions for the Verbal Comprehension Index (VCI), and Working Memory Index (WMI) than for other regions of the brain (Coalson et al., 2010). While not perfect, researchers continue to work on reducing the flaws and mistakes that these examinations make so that IQ testing can continue to act as a dependable tool for intelligence testing and research.

References

1. Coalson, D. L., Raiford, S. E., Saklofske, D. H., & Weiss, L. G. (2010). WAIS-IV. *WAIS-IV Clinical Use and Interpretation*, 3–23. https://doi.org/10.1016/b978-0-12-375035-8.10001-1

2. Colom, R., Karama, S., Jung, R. E., & Haier, R. J. (2010). Human intelligence and brain networks. *Dialogues in Clinical Neuroscience*, *12*(4), 489–501. https://doi.org/10.31887/dcns.2010.12.4/rcolom

3. Dechaume, M., Mercier, M., Feybesse, C., Lubart, T., Chouvelon, G., Kermarrec, S., & Tordjman, S. (2024). Is intelligence necessary and sufficient for creativity? an analysis of convergent and divergent thinking. *Learning and Individual Differences*, *116*, 102575. https://doi.org/10.1016/j.lindif.2024.102575

4. Goriounova, N. A., & Mansvelder, H. D. (2019, February 15). *Genes, cells and brain areas of intelligence*. Frontiers in human neuroscience. https://pmc.ncbi.nlm.nih.gov/articles/PMC6384251/

5. Koriakin, T. A., Mccurdy, M. D., Papazoglou, A., Pritchard, A. E., Zabel, T. A., Mahone, E. M., & Jacobson, L. A. (2013). Classification of intellectual disability using the wechsler intelligence scale for children: Full scale iq or general abilities index? Developmental Medicine & Child Neurology, 55(9), 840–845. https://doi.org/10.1111/dmcn.1220

6. Iida, Y., Chiba, H., Ikeda, S., Tohyama, H., & Ikoma, K. (2021). Association between the Wechsler Adult Intelligence Scale III and early return to work after Traumatic Brain Injury. Work, 68(4), 1101–1111. https://doi.org/10.3233/wor-213440

7. Loring DW, Bauer RM. Testing the limits: Cautions and concerns regarding the new Wechsler IQ and Memory scales. Neurology. 2010;74(8):685-690. doi:10.1212/WNL.0b013e3181d0cd12

8. Niileksela, C. R., & Reynolds, M. R. (2019). Enduring the tests of age and time: Wechsler constructs across versions and revisions. Intelligence, 77, 101403. https://doi.org/10.1016/j.intell.2019.101403

9. Reynolds, C. R., Altmann, R. A., & Allen, D. N. (2021). The problem of bias in psychological assessment. Mastering Modern Psychological Testing, 573–613. https://doi.org/10.1007/978-3-030-59455-8_15

10. SFU. (2005). Intelligence Theories. Learner differences. https://www.sfu.ca/~jcnesbit/EDUC220/week4/week4.html

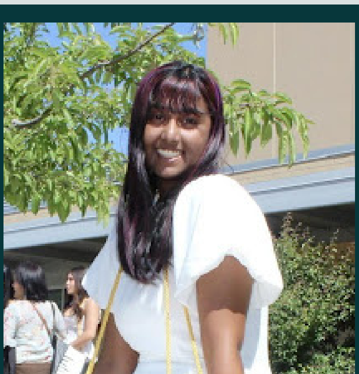
11. Sternberg, R. J. (2012). Intelligence. Dialogues in Clinical Neuroscience, 14(1), 19–27. https://doi.org/10.31887/dcns.2012.14.1/rsternberg

12. Sternberg, R. J., & Jarvin, L. (2015). Binet, Alfred (1857–1911). International Encyclopedia of the Social & Behavioral Sciences, 596–599. https://doi.org/10.1016/b978-0-08-097086-8.61008-5

13. Warne, R. T., Burton, J. Z., Gibbons, A., & Melendez, D. A. (2019). Stephen Jay Gould's analysis of the Army beta test in the mismeasure of man: Distortions and misconceptions regarding a pioneering mental test. Journal of Intelligence, 7(1), 6. https://doi.org/10.3390/jintelligence7010006

14. Watkins, M. W., & Smith, L. G. (2013). Long-term stability of the Wechsler Intelligence Scale for children—Fourth edition. Psychological Assessment, 25(2), 477–483. https://doi.org/10.1037/a0031653

15. Worhach, J., Boduch, M., Zhang, B., & Maski, K. (2021). Remote assessment of pediatric patients with daytime sleepiness and healthy controls: A pilot study of feasibility and reliability. Child Neurology Open, 8. https://doi.org/10.1177/2329048x211048064

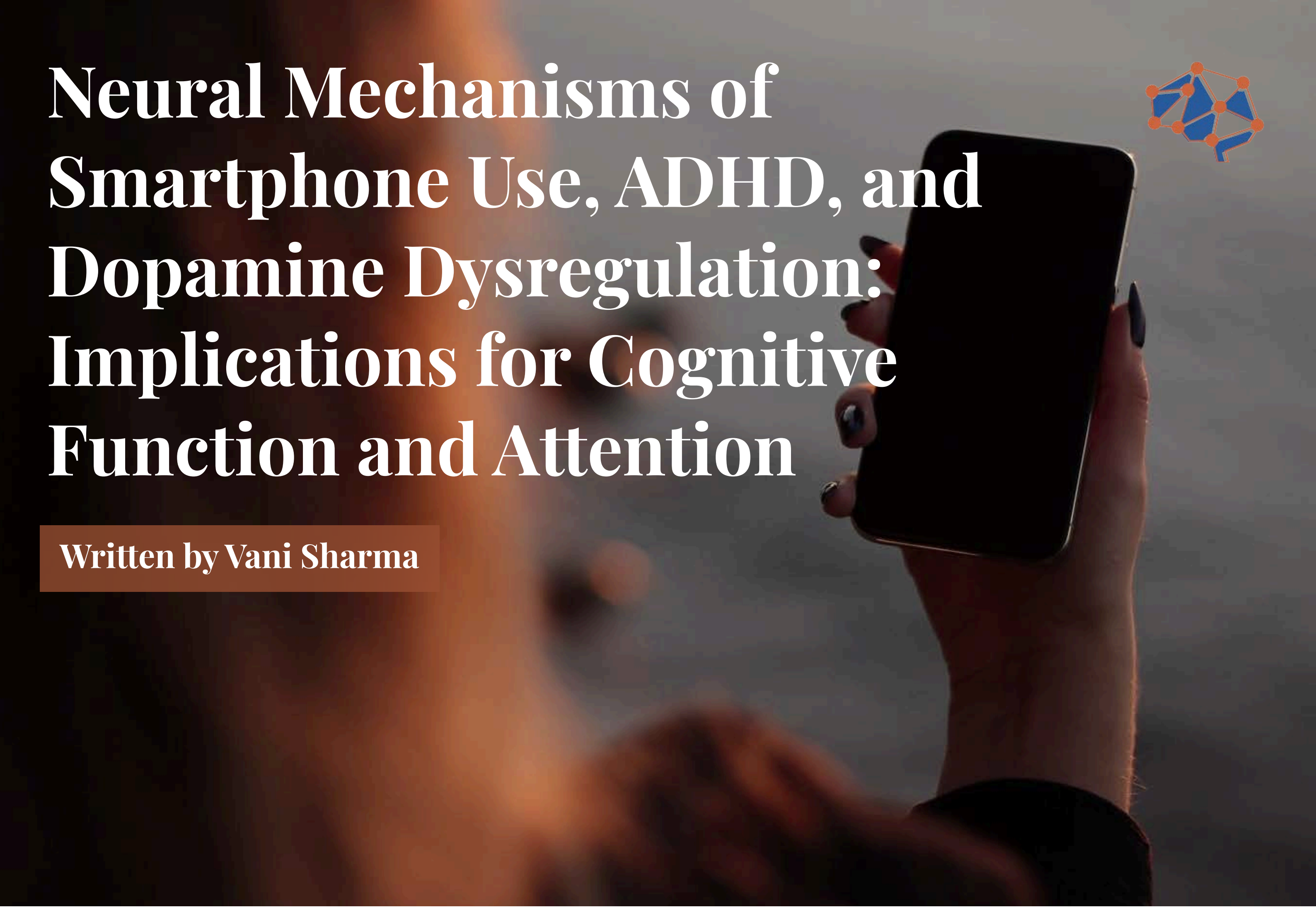


About the Author

Ananya Sampathkumar is a sophomore, majoring in Neuroscience with minors in Chemistry and Public Health. Outside of Brain Matters, Ananya is an assistant editor-in-chief for Double Helix Digest, a member of Starcourse, a volunteer at Carle Hospital, and works at the Office of Undergraduate Admissions as a tour guide and student ambassador. In her free time, Ananya likes to read books, make jewelry, watch movies, and hang out with her friends.

Neural Mechanisms of Smartphone Use, ADHD, and Dopamine Dysregulation: Implications for Cognitive Function and Attention

Written by Vani Sharma



Introduction

The pervasiveness of smartphones in contemporary society has redefined human cognition, affecting attention, memory, and choice. At the core of this interaction is the brain's dopaminergic system, which underlies reward processing and reinforcement learning. Dopamine release, triggered by random digital stimuli in the form of notifications and social media feedback, creates a cycle of compulsive phone use, akin to behavioral reinforcement processes in addictive disorders foreshadowing an extreme impact on the dopaminergic system of the brain.. Meanwhile, ADHD is characterized by dysregulated dopamine signaling in key neural circuits, including the prefrontal cortex (PFC), striatum, and midbrain structures.

The neurobiological convergence of ADHD and problematic smartphone use indicates that attention-deficit individuals might be especially susceptible to technology overuse. This article discusses the interaction between dopamine dysregulation caused by smartphone use and neural mechanisms of ADHD, with special reference to cognitive functioning and attentional control. Elucidating these interactions can provide insight into the impact of contemporary technology on cognitive functioning and guide interventions to prevent its possible negative effects.

Dopamine, Reward Circuitry, and Smartphone Use

Dopamine plays a fundamental role in the brain reward system, where it primarily controls neuronal activity in the mesocorticolimbic pathway, which includes the ventral tegmental area (VTA), nucleus accumbens (NAc), and PFC (Volkow et al., 2018). The system is responsible for reinforcing behavior that has pleasurable effects, driving motivated and habitual behavior. Smartphone usage, particularly that based on social media and notifications, activates this pathway by offering intermittent rewards and fueling habitual checking behaviors (Montag et al., 2019).

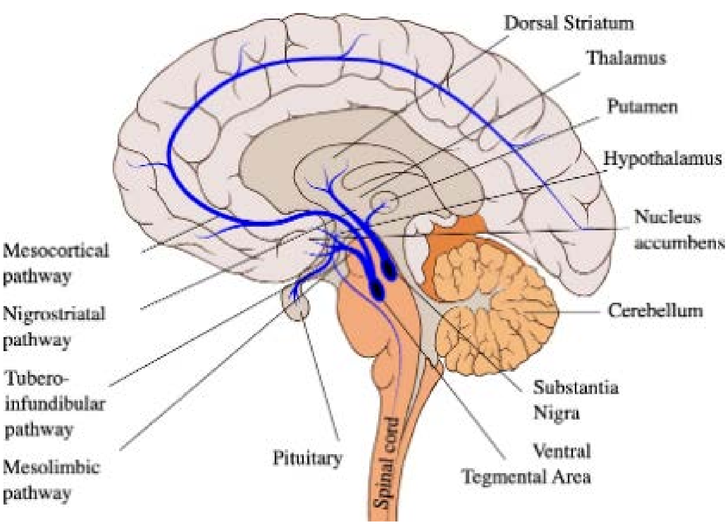


Figure 1. Dopaminergic Pathways & Crude Neuroanatomy (Wikimedia Commons)

Similar to substance use disorders, excessive phone use can lead to the downregulation of dopamine receptors, decreasing the brain's sensitivity to natural rewards and reinforcing compulsive use behaviors (Kühn & Gallinat, 2015). The repeated overstimulation of these circuits can regulate synaptic plasticity, which is expressed as attentional impairment as well as cognitive impulsivity in individuals susceptible to ADHD.

Neuroanatomical Foundations of Attention Dysregulation

ADHD and chronic smartphone use have been correlated with structural as well as functional changes within brain regions crucial for attentional control. Neuroimaging studies show reduced gray matter volume in the PFC and ACC in ADHD individuals, interrupting executive function and impulse regulation (Shaw et al., 2007). Excessive screen time exerts a similar effect on these regions, with prolonged digital exposure linked to reduced functional connectivity between the PFC and striatum, the hallmark of impaired top-down cognitive control (Firth et al., 2019). The interference of dopamine signaling within the frontostriatal circuits, which is shared by both ADHD and addictive phone use, therefore reflects a shared neural process that may additionally strengthen attentional difficulties along with cognitive instability in the individuals concerned.

Behavioral and Neurological Implications of Dopamine Overload

The chronic hyperstimulation of the brain's reward system through smartphone usage may lead to fundamental alterations in attentional processing as well as self-regulation. Research indicates that individuals with high smartphone dependency have reduced attentional blink capacity and working memory impairment, due to overstimulation of dopaminergic activation and neural fatigue in attention networks (Loh & Kanai, 2016). Additionally, compulsive reinforcement of online behavior has the potential to interfere with the brain's ability to sustain focus on tasks requiring deep cognitive processing, a particularly troublesome situation for those with ADHD (Wilmer et al., 2017). Promising treatments include behavioral modification in the form of digital detoxification practices and enforced screen time limitations, intending to restore dopamine homeostasis and re-establishing attentional control.

Conclusion

This intersection of smartphone behavior, dopamine dysregulation, and ADHD constitutes a central axis of concern within modern cognitive neurosciences. The addictive power of digital messaging, driven by intermittent reward and dopamine release, demonstrates the attention disorder neurobiology. With associated structural and functional brain changes present in both heavy phone use and ADHD, ongoing research is critical to define chronic digital consumption effects on the brain over the long term. However, precautions can be taken beforehand to mitigate these effects and preserve attentional integrity in an increasingly digital world.

> **Strategies such as mindful use of technology—taking scheduled phone breaks, disabling notifications, or employing grayscale mode to reduce visual salience—can alleviate compulsive engagement.**

Cognitive training through attentional control exercises, meditation, or working memory training can also improve executive function and digital distraction resistance. Behavioral therapies, including screen-time monitoring apps, dopamine fasting, and replacing digital activity with offline hobbies, offer other means of controlling excessive phone usage. Environmental adjustments, such as establishing tech-free zones, keeping phones out of the workplace, and using physical alarm clocks instead of smartphones, can also facilitate improved habits. By combining these strategies, individuals can offset the neural effects of long-term smartphone use, decreasing the likelihood of attentional deficits and promoting higher cognitive control in a world where digital stimuli prevail.

References

1. File: Ventral tegmental area.svg. (2022, August 2). Wikimedia Commons. Retrieved 17:36, April 2, 2025 From https://commons.wikimedia.org/w/index.php?title=File:Ventral_tegmental_area.svg&oldid=679710867.

2. Firth, J., Torous, J., Stubbs, B., Firth, J. A., Steiner, G. Z., Smith, L., & Sarris, J. (2019). The neural basis of digital distraction and its cognitive consequences: A systematic review and meta-analysis. *Neuroscience & Biobehavioral Reviews*, 105, 12-21.

3. Individual Care of Texas. (2024). *Digital Detox for Mental Clarity: A Guide to Reclaim Focus*. Google image result for https://individualcareoftx.com/wp-content/uploads/2024/01/blog-highlands-jan-1.jpg. https://images.app.goo.gl/iAkP7d3Sg24L8YWH8

4. Kühn, S., & Gallinat, J. (2015). Brains online: Structural and functional correlates of habitual Internet use. *Addiction Biology*, 20(2), 415-422.

5. Loh, K. K., & Kanai, R. (2016). How has the Internet reshaped human cognition? Neuroscientist, 22(5), 506-520.
6. Montag, C., Lachmann, B., Herrlich, M., & Zweig, K. (2019). Addictive features of social media/messenger platforms and Freemium games against the background of psychological and economic theories. *International Journal of Environmental Research and Public Health*, 16(14), 2612.
7. Shaw, P., Eckstrand, K., Sharp, W., Blumenthal, J., Lerch, J. P., Greenstein, D., Clasen, L., Evans, A., Giedd, J., & Rapoport, J. (2007). Attention-deficit/hyperactivity disorder is marked by slowing of cortical maturation. *Proceedings of the National Academy of Sciences*, 104(49), 19649-19654.
8. Volkow, N. D., Wang, G. J., Tomasi, D., & Baler, R. D. (2018). The neuroscience of addiction: Implications for clinical practice. *Nature Reviews Neuroscience*, 19(4), 224-236.

About the Author

Vani Sharma is pursuing a Bachelor of Science in Molecular and Cellular Biology (MCB) with an honors concentration, alongside a minor in Public Health and a Neuroscience certificate. As a writer for Brain Matters, she investigates the intricate interplay between the brain and diverse phenomena, including the neural foundations of gratitude, the influence of music on cognitive processes, and the complexities of neuroanatomy and neurological disorders. Through her work, she blends rigorous scientific research with engaging narratives to illuminate the brain's extraordinary intricacies while promoting scientific literacy and making complex concepts accessible to a broader audience.

The Effects of Physical Exercise on Memory and Cognitive Functions

Written by Navi Singh

Cognitive functions are the mental processes that allow us to think, learn, and focus. Memory, in particular, involves the encoding, storage, and retrieval of information—processes that are essential for daily activities. Emerging evidence in neuroscience research points to a strong relationship between physical health and cognitive performance. For instance, a study by Donnelly et al. highlights a strong link between regular physical exercise and improved cognitive health (Donnely, 2016). Particularly, studies show that individuals who engage in physical activity demonstrate better memory, attention, and problem-solving abilities compared to those who do not exercise. These findings are particularly relevant in the context of neurodegenerative diseases such as Alzheimer's and dementia, which involve a progressive loss of brain structure and function and significantly impair cognitive abilities such as memory. Examining the impact of physical activity on memory and cognition, and summarizing recent literature on its role in the development or prevention of neurodegenerative diseases, is therefore essential for guiding future neuroscience research.

Physical activity has been shown in several studies to reduce the risk of neurological diseases such as Alzheimer's and dementia. One of the key ways exercising benefits the brain is by enhancing brain activity through reduction of inflammation, which could impact cognitive impairment. One central mechanism involved in the regulation is brain-derived neurotrophic factor (BDNF), a protein that plays a crucial role in supporting neuronal survival, growth, and synaptic plasticity. BDNF is important for long-term potentiation, a cellular process that is essential to help with learning and memory formation, promoting the strength of connections between neurons. Exercising stimulates BDNF expression in the hippocampal pathway, which can improve memory. A recent study by Sanaeifar et. al. found that individuals who regularly engage in aerobic exercise exhibited increased levels of BDNF and reduced levels of neuroinflammation, overall improving cognitive function. (Sanaeifar, 2024).

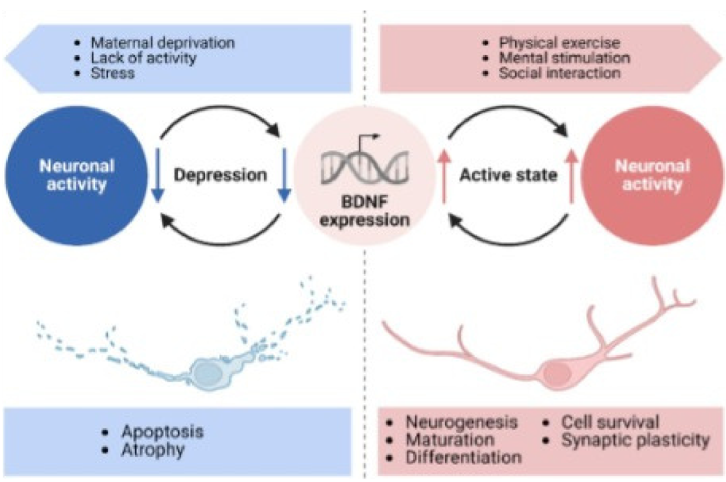


Figure 1. The impact of BDNF expression on neuronal activity in individuals who exercise and those who do not exercise (Sanaeifar, 2024)

Additionally, when individuals age, neurogenesis and synaptic plasticity naturally decline. However, studies have shown that individuals who regularly exercise maintain these essential mechanisms and preserve cognitive function at an older age. For example, a research article by Iso-Marrku demonstrated that physically active older adults had a lower risk of developing neurodegenerative diseases such as Alzheimer's and dementia. (Iso-Markku, 2022; Lopez-Ortiz, 2023). These findings show the importance of exercising daily to preserve cognitive function and prevent early-onset neurodegenerative diseases.

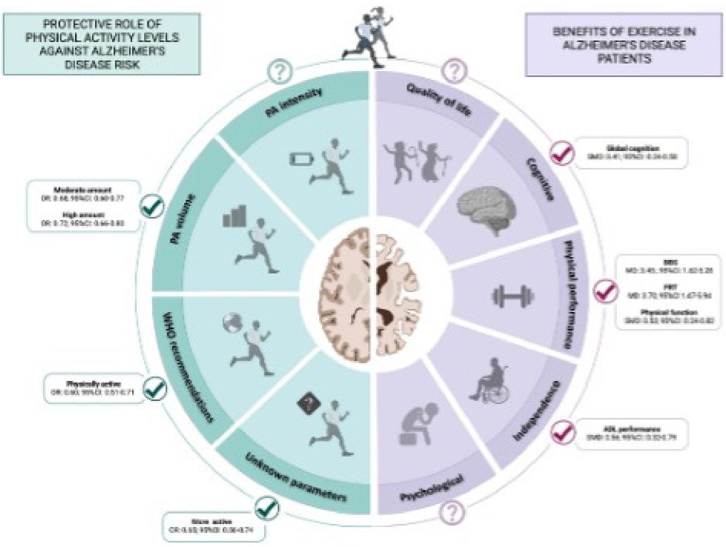


Figure 2. Benefits of exercise in prevention of Alzheimer's disease (Lopez-Ortiz, 2023)

Not only can exercise prevent neurodegenerative disease, but it also improves memory, attention, and overall cognitive activity. Research has shown that both short-term and long-term exercise can lead to cognitive improvements. Short-term exercises, like aerobic exercise, have been found to enhance working memory and attention by increasing the cerebral blood flow to the brain. Long-term exercise, on the other hand, can cause functional changes in the brain, like increasing hippocampal volume, which is an area of the brain crucial for memory processing. (Donnelly, 2016). In a review by Donnelly et. al., the authors highlighted that consistent physical activity is associated with improvements in cognition, memory, and executive function. Exercise interventions mentioned in Donnelly's review can lead to enhanced academic performance and cognitive development for adults and children. Moreover, different types of exercise have been found to impact cognition uniquely. Aerobic exercises like running have been shown to see improvements in memory and attention, whereas resistance training has been associated with better working memory and executive function. Even yoga and meditation have been shown to provide cognitive benefits in reducing stress and improving attention. Altogether, these findings suggest that exercise has a multitude of benefits:

> “Not only does it prevent cognitive decline, but it also reduces inflammation in the brain, increases hippocampal volume, and optimizes cognitive function at all ages (Voss, 2023).”

In conclusion, the evidence in the current neuroscience literature strongly supports that physical exercise has been shown to enhance memory and cognitive function and also reduce the risk of neurodegenerative diseases like Alzheimer's and dementia. Biological mechanisms, such as increased BDNF levels and reduced neuroinflammation, improve the memory and function of the hippocampus. Additionally, regular physical activity, whether short-term or long-term, preserves cognitive ability as individuals get older and prevents the onset of Alzheimer's and dementia. Any type of exercise plays a crucial role in improving memory and attention. However, there are limitations to research regarding the long-term effects of different exercise models. Future studies will show what specific types of long-term exercise are crucial and bring the most cognitive benefits. Understanding the importance of the impact of physical health on the brain can lead to more targeted interventions to help reduce the risk of cognitive decline and improve attention and memory.

References

1. Donnelly, J. E., Hillman, C. H., Castelli, D., Etnier, J. L., Lee, S., Tomporowski, P., Lambourne, K., & Szabo-Reed, A. N. (2016). Physical Activity, Fitness, Cognitive Function, and Academic Achievement in Children: A Systematic Review. *Medicine and science in sports and exercise, 48*(6), 1197–1222. https://doi.org/10.1249/MSS.0000000000000901

2. Iso-Markku, P., Kujala, U. M., Knittle, K., Polet, J., Vuoksimaa, E., & Waller, K. (2022). Physical activity as a protective factor for dementia and Alzheimer's disease: systematic review, meta-analysis and quality assessment of cohort and case-control studies. *British journal of sports medicine, 56*(12), 701–709. https://doi.org/10.1136/bjsports-2021-104981

3. López-Ortiz, S., Lista, S., Valenzuela, P. L., Pinto-Fraga, J., Carmona, R., Caraci, F., Caruso, G., Toschi, N., Emanuele, E., Gabelle, A., Nisticò, R., Garaci, F., Lucia, A., & Santos-Lozano, A. (2023). Effects of physical activity and exercise interventions on Alzheimer's disease: an umbrella review of existing meta-analyses. *Journal of neurology, 270*(2), 711–725. https://doi.org/10.1007/s00415-022-11454-8

4. Sanaeifar, F., Pourranjbar, S., Pourranjbar, M., Ramezani, S., Mehr, S. R., Wadan, A. S., & Khazeifard, F. (2024). Beneficial effects of physical exercise on cognitive-behavioral impairments and brain-derived neurotrophic factor alteration in the limbic system induced by neurodegeneration. *Experimental gerontology, 195*, 112539. https://doi.org/10.1016/j.exger.2024.112539
5. Voss, S., Cerna, J., & Gothe, N. P. (2023). Yoga Impacts Cognitive Health: Neurophysiological Changes and Stress Regulation Mechanisms. *Exercise and sport sciences reviews, 51*(2), 73–81. https://doi.org/10.1249/JES.0000000000000311

About the Author

Navi Singh is a rising Junior at the University of Illinois Urbana-Champaign, majoring in Neuroscience and minoring in Health Administration and Chemistry on the Pre-Medical track. She is an aspiring physician hoping to specialize in Neurology. She is also involved with several RSO's on campus. These include serving as Social Chair for Udaan and the Undergraduate Neuroscience Society, being a member of a Pre-Health Professional Fraternity Phi Chi, mentor for Illini Mentor Program, volunteer for Global Medical Brigades, and Research Assistant at the Kukekova lab. Navi is excited to share her first article and research with Brain Matters!

Survival vs. Cognition: Stress Mechanisms in Humans vs. Animals

Written by Pravika Srivastava

Introduction to Stress Mechanisms

Stress is defined as the physiological and psychological reaction to perceived threats or demands that activates a cascade of neurobiological processes that help maintain homeostasis (McEwen, 2007). The hypothalamic-pituitary-adrenal axis (The HPA Axis) plays the primary role in regulating stress and the release of stress hormones such as cortisol and corticosterone. These hormones are used to regulate the physiological and behavioral responses in several species (Sapolsky, Romero, & Munck, 2000). Additionally, neurotransmitters play an equally important role in the regulation of stress and emotion. Specifically, dopamine and norepinephrine influence stress-related cognitive and emotional regulation (Joëls & Baram, 2009). Even with similar processes of regulating stress, animals and humans tend to respond to and process stress differently. Animals are primarily concerned with acute survival-linked stressors, such as predation or resource deficiency. Whereas, humans experience complex cognitive appraisal, differentiating between immediate threats and external concerns like societal or financial pressures. It is crucial to highlight and study these differences between human and animal stress responses as it allows us to improve the understanding of chronic stress disorders and coping strategies in humans while also enhancing animal stress research.

Fight-or-Flight Response in Humans vs. Animals

Animals and humans both respond differently to perceived threats; however, one mechanism that allows both to respond rapidly is the fight-or-flight response. This response is quite instinctive in animals and is triggered by the autonomic nervous system, especially in the sympathetic branch. This area is known to initiate rapid physiological changes like increased heart rate, energy mobilization and pupil dilation (Cleveland Clinic, 2022). In animals, these reactions and changes are crucial for survival during acute stressors. These stressors entail predator-prey encounters, where quick action is needed and can determine life or death. The HPA axis releases glucocorticoids that help maintain energy levels and modulate inflammation during high-stress periods such as these (Wingfield & Romero, 2001). However, in humans, physiological responses also have an added cognitive aspect. Humans tend to interpret stress through fear and anxiety and this is based on whether the stress is a direct and immediate stressor or an anticipated one. Since humans can overthink problems, it is common for humans to stay stressed even after the direct stressor has left. Unlike animals, we continue to worry about things that have not even occurred or happened in our work and social lives. This tendency makes it more difficult for humans to recover and overtime leads to issues such as anxiety and burnout (Harvard Health Publishing, 2018).

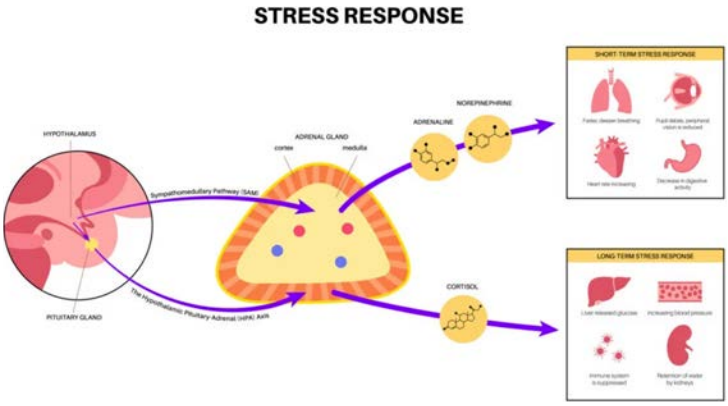


Figure 1. Diagram showing the human stress response pathways. The SAM pathway triggers immediate release of adrenaline and norepinephrine (fight-or-flight), while the HPA axis leads to cortisol release during prolonged stress. *Simply Psychology, "What is the HPA Axis?"*

Survival-Based Stress Response in Humans vs. Animals

Stress in animals is highly survival-driven and is elicited by immediate threats by predators, famine or weather. These acute stressors can trigger fast physiological adjustments that enable them to hide, fight or even run away. These reactions have been perfected over the course of evolution to maximize survival (Wingfield & Romero, 2001). As the danger leaves, the stress response switches off, and the animal can recover by returning to rest and conserving energy. Figure 2 illustrates this process, showing how animals pass through an ordered cascade of physiological and behavioral responses to perceived danger, and recovery or chronic stress outcome in relation to their capacity to recover homeostasis. The way that animals are able to recover so quickly is a significant evolutionary adaptation that saves animals from experiencing effects of chronic stress. On the other hand, humans perceive stress in addition to physical danger. Some examples could include public speaking, issues at work and in relationships. This difference between humans and animals is because humans have an advanced cognitive ability to imagine, evaluate and magnify risks (Ohman, 2005.) Due to this, humans often feel stressed in response to psychological or social problems in the absence of any real life-threatening situation. This is beneficial as humans can adapt their actions allowing them to prepare and evade threats, but it also leads to chronic disease (Harvard Health Publishing, 2018).

Long-Term Stress: Chronic Stress in Animals and Humans

Animals in the wild are oftentimes subjected to brief, severe stressors like predators or harsh weather. These difficulties trigger quick physiological reactions, such as the release of glucocorticoids, which minimize long-term harm by rapidly returning to baseline after the threat has passed (Wingfield and Romero, 2001). However, some animals are kept in

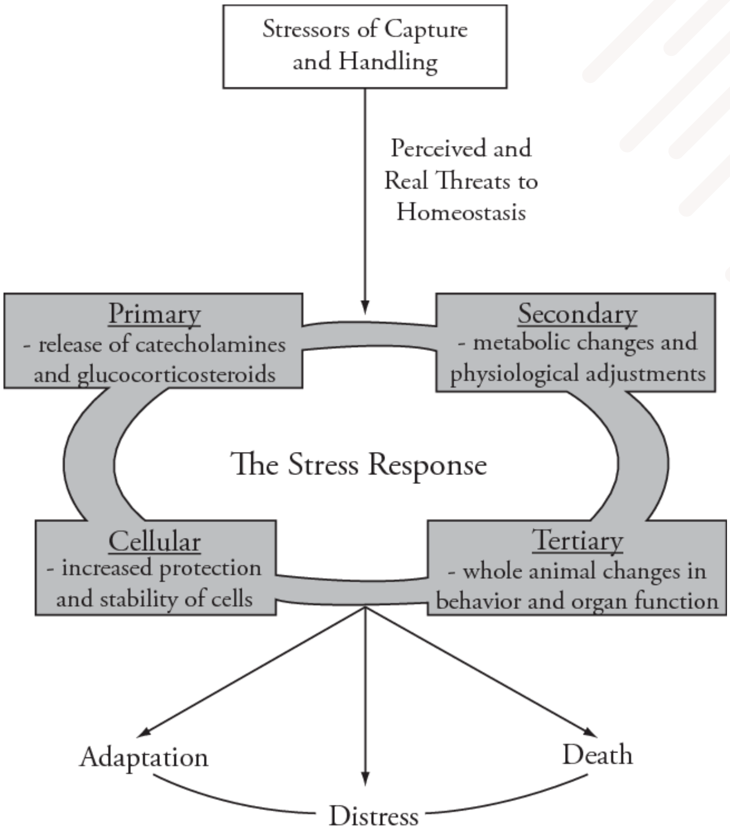


Figure 2. Powell, Roger A., et al. "Diagram illustrating the stress response that follows when an animal perceives a threat to homeostasis." *ResearchGate*, 2012

captivity which can lead to ongoing stress. This is due to confinement, lack of stimulation, and abnormal social structures. This is due to confinement, lack of stimulation, and abnormal social structures. According to Morgan and Tromborg, these disorders may cause a persistent increase in stress hormones, which can have a detrimental effect on immune system performance, reproductive success, and general animal behavior. Long-term exposure to glucocorticoids in captive animals has led to chronic stress. Behaviors such as disruption in circadian rhythms, and impairment of hippocampus function are just some signs (McEwen, 2007). In people, psychological and social triggers like employment obligations, interpersonal issues, and unstable finances are more frequently the cause of chronic stress. Given that humans have a more developed prefrontal cortex as opposed to animals, they are able to predict, consider, and magnify stressors, triggering the hypothalamic-pituitary-adrenal (HPA) axis to continuously activate (Joëls and Baram, 2009). Eventually, this imbalance leads to a variety of illnesses, such as diminished immune system, anxiety, depression, and cardiovascular disease (McEwen; Chrousos and Gold, 1998). The frequency of exposure can lead to long-term physiological damage is explained by the idea of allostatic load, which is the overall deterioration on the body brought on by chronic stress (McEwen and Seeman, 1999). These consequences highlight the importance of resilience-building techniques and stress-reduction tactics in human health care and lifestyle.

Support Systems and Growth Mindsets

Though its structure and purpose vary among different species, social support is crucial for controlling stress in both people and animals. Stress levels in animal communities can be very much influenced by social hierarchies and group dynamics. For instance, constant social stress—especially in captivity—allows subordinate animals within rigid hierarchies to have higher glucocorticoid levels. On the other hand, animals that create stable social ties—such as grooming-engaging primates—often have reduced levels of stress hormones, underscoring the protective effect of positive social interactions (Wingfield and Romero, 2001). In humans, networks of social support—including family, friends, and community members—act as shielding buffers against ongoing psychological stress.These interactions help to downregulate the activity of the sympathetic nervous system and the HPA axis, thus reducing the physiological consequences of stress (McEwen, 2007). Besides, resilience against stress depends on a person's psychological viewpoint.

> "The ability to reframe stress through a growth mindset helps humans to see obstacles as opportunities rather than hazards."

This cognitive assessment modulates the stress reaction by influencing the way the brain understands threats and their consequent physiological effects (Joëls and Baram, 2009). Using adaptive coping techniques that support emotional control and lower allostatic load—cognitive reappraisal, mindfulness, and goal-setting—helps one to reduce persistent stress (McEwen and Seeman, 1999). Consequently, how different animals react to and recover from stress depends much on both internal mental frameworks and outside social settings.

Implications for Behavioral Models and Future Research:

This paper looked into how human and animal stress mechanisms differ, emphasizing how humans frequently experience cognitively driven, chronic stress while animals depend on acute, survival-based reactions. Both make use of comparable biological systems, such as the HPA axis and the sympathetic nervous system, but because of our superior cognitive abilities, humans are exposed to stress for longer periods of time and experience more complicated effects. Important differences have been observed in the influence of social and psychological factors, long-term stress development, and fight-or-flight reactions. Behavioral psychology greatly benefits from the research of stress processes in human-animal relationships. Stress reactions have a direct impact on behavior, decision-making, and emotional regulation since they are based on common neurobiological systems such as the sympathetic nervous system and the HPA axis (Joëls and Baram, 2009). More specialized behavioral models, like those for anxiety, depression, and post-traumatic stress disorder (PTSD), can be guided by knowledge of how long-term stress changes the brain circuits involved in cognition and emotion. For instance, knowledge gained from research on animals has influenced pharmacological and exposure-based therapies to control the release of stress hormones and brain plasticity (McEwen, 2007). But the primary issue is the morality of using animals in stress studies. In order to protect animal welfare while advancing science, researchers must provide humane conditions, reduce suffering, and use alternatives when possible (Morgan and Tromborg, 2005). The relationship between stress resilience, neuroplasticity, and specialized treatment approaches must be further investigated in future studies. To better understand how early life stress, social context, and psychological state interact to influence long-term health outcomes, more longitudinal research in humans is needed. Public health and tailored treatment can also benefit from studying how stress appears in other animals and social groups. Combining psychological ideas with neurobiological facts may also improve our capacity to anticipate stress vulnerability and stop it from happening. Future research can better understand stress problems and inform more efficient, moral, and focused treatments by integrating insights from animal studies into human behavioral science.

References

1. AF;, A. (n.d.). Stress signalling pathways that impair prefrontal cortex structure and function. *Nature reviews. Neuroscience*. https://pubmed.ncbi.nlm.nih.gov/19455173/

2. Arne Öhman, Summary Behavioral data suggest that fear stimuli automatically activate fear and capture attention. This effect is likely to be mediated by a subcortical brain network centered on the amygdala. Consistent with this view, Vuilleumier, P., Anderson, A. K., Armony, J. L., Bishop, S. J., Carlsson, K., Cunningham, W. A., Davidson, R. J., Dimberg, U., Globisch, J., LeDoux, J. E., Miltner, W. H. R., & Morris, J. S. (2005a, June 16). The role of the amygdala in human fear: Automatic detection of threat. *Psychoneuroendocrinology*. https://www.sciencedirect.com/science/article/abs/pii/S0306453005001022

3. on, P. by P. M. (2022, April 11). Human steam. *Human STEAM*. https://humanap.community.uaf.edu/2022/04/11/hpa-axis-effects-of-stress%EF%BF%BC/

4. Understanding the stress response. *Harvard Health.* (2024, April 3). https://www.health.harvard.edu/staying-healthy/understanding-the-stress-response

5. Joëls, M., & Baram, T. Z. (2009, April 2). The neuro-symphony of stress. *Nature News.* https://www.nature.com/articles/nrn2632

6. BS;, M. (n.d.). Physiology and neurobiology of stress and adaptation: Central Role of the brain. *Physiological reviews.* https://pubmed.ncbi.nlm.nih.gov/17615391/

7. professional, C. C. medical. (2025, April 1). Sympathetic nervous system (SNS): What it is & function. *Cleveland Clinic.* https://my.clevelandclinic.org/health/body/23262-sympathetic-nervous-system-sns-fight-or-flight

8. Rosmond, R., Dallman, M. F., & Björntorp, P. (1998). Stress-related cortisol secretion in men: relationships with abdominal obesity and endocrine, metabolic and hemodynamic abnormalities. *The Journal of Clinical Endocrinology & Metabolism, 83*(6), 1853-1859.

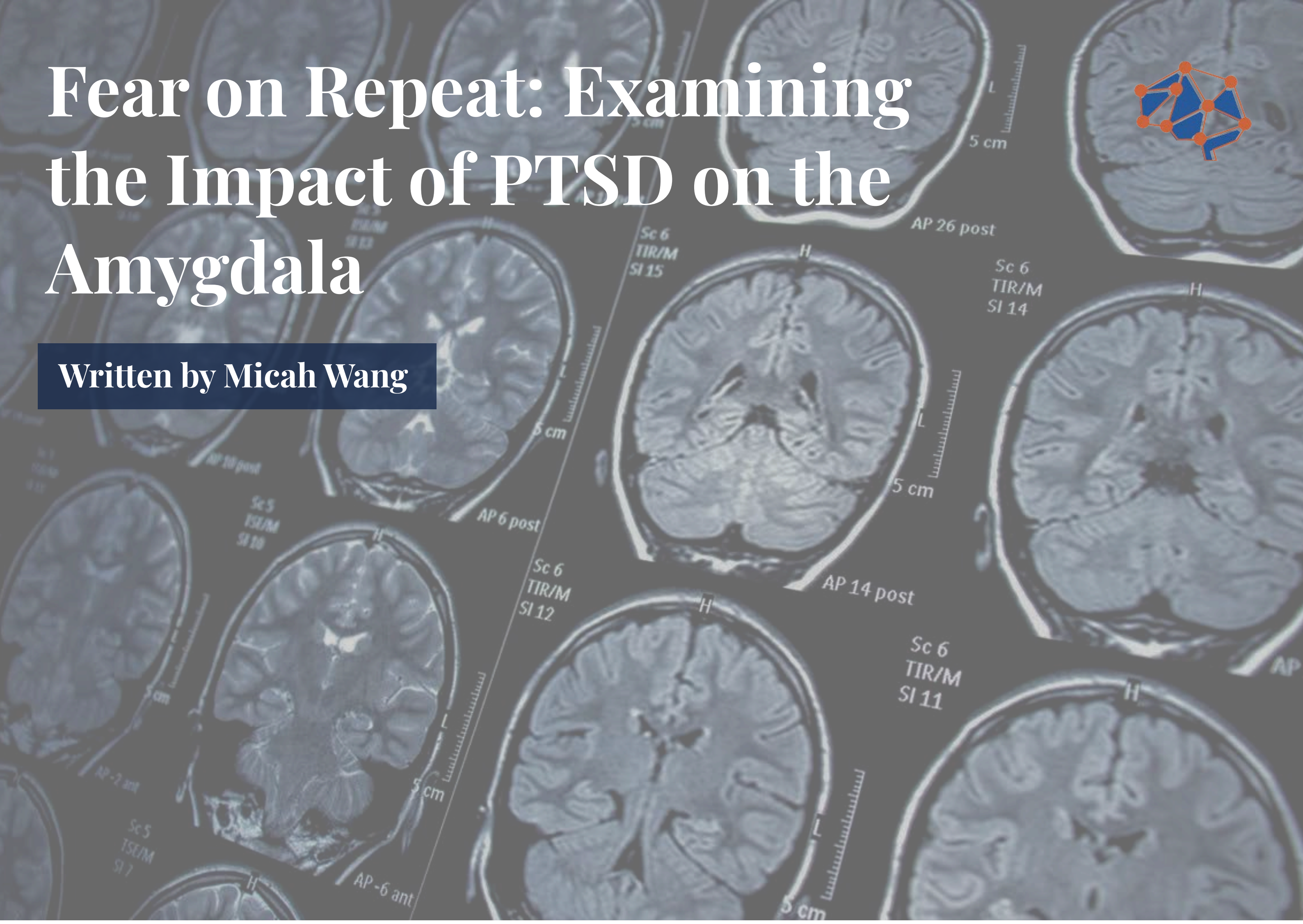
9. Sapolsky, R. M., Romero, L. M., & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine reviews, 21*(1), 55-89.

10. Williams, K. (2023). Cat in Repose. Island in the Net. Retrieved June 2, 2025, from https://islandinthenet.com/cat-in-repose/

11. 2 diagram illustrating the stress response that follows ... (n.d.-a). https://www.researchgate.net/figure/Diagram-illustrating-the-stress-response-that-follows-when-an-animal-perceives-a-threat_fig1_248706927

About the Author

Pravika Srivastava is a rising junior at the University of Illinois Urbana-Champaign majoring in neuroscience with a minor in psychology on the pre-medical track. She is passionate about brain health, mental well-being, and hopes to pursue a career in psychiatry. As a writer and new Social Media Co-Chair for Brain Matters, she enjoys writing about neuroscience-related topics while eager in helping expand the journal's outreach. On campus, Pravika volunteers in the Pediatric ICU at Carle Foundation Hospital, conducts research at the CONNECTlab and Rudolph Lab, and serves on the Speaker Committee for Alpha Epsilon Delta. Pravika is excited to share her research and writing as part of her ongoing commitment to advancing understanding of the brain and mental health.



Fear on Repeat: Examining the Impact of PTSD on the Amygdala

Written by Micah Wang

What is PTSD?

Post-traumatic stress disorder (PTSD) is a psychiatric disorder inflicted by experiencing or witnessing a traumatic event, triggering a variety of symptoms. Individuals with PTSD can experience symptoms such as nightmares, flashbacks, and detached behavior, leading to the inability to function normally, particularly in social or family life (Iribarren et al., 2005). These symptoms can affect a person for a lifetime, emphasizing the importance of this disorder.

What is the amygdala?

The amygdala is one of the main brain regions that is affected by PTSD. The amygdala is a cluster of nuclei that lies in front of the hippocampus and near the temporal lobe (Johns, 2016). The amygdala is divided into many different sections of nuclei, but the three main groups are the basolateral, corticomedial, and the central nucleus (Johns, 2016). The basolateral group receives visual and auditory projections from the temporal lobe, and the corticomedial group receives input from the olfactory bulb, which processes smell information. Therefore, the corticomedial group is more important in animals with a keen sense of smell. The central nucleus elicits emotional responses and projects them to the hypothalamus and autonomic region of the brain stem (Johns, 2016). In a study where lesions of the central nucleus were found, fear conditioned responses were eliminated, which suggests its involvement in experiencing fear (Ressler, 2010).

The amygdala processes external stimuli as well as regulatory stimuli that come from connections with areas of the brain that modulate the amygdala (Ressler, 2010). Areas such as the prefrontal cortex and the sensory cortical and thalamic areas mediate subregions in the amygdala and are involved in inhibiting its ability to elicit fear responses (Ressler, 2010).

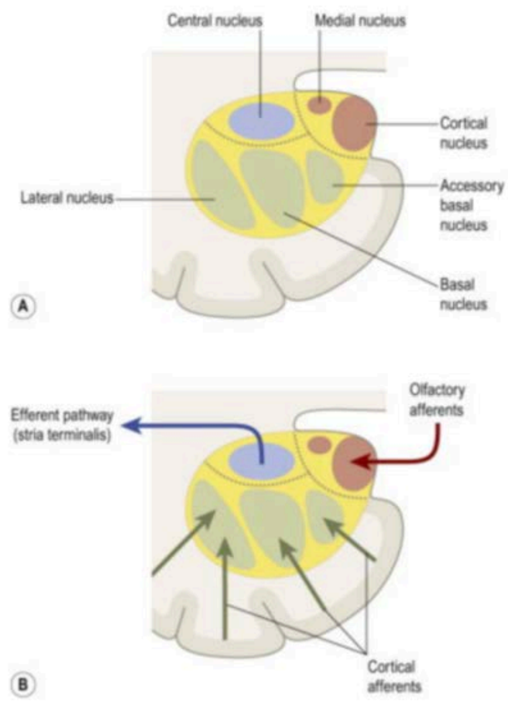


Figure 1. A) Diagram of the amygdala and its subregions. **B)** Central nucleus is involved in sending response output via the stria terminalis, which is a major output pathway for the amygdala. The cortical nucleus receives olfactory stimuli while the basal nucleus receives non-olfactory stimuli.

How does PTSD affect the amygdala?

The amygdala is one of the most strongly involved brain structures in the pathophysiology of PTSD (Morey et al., 2012). However, studies that have shown differences in amygdala volume in those with PTSD fail to draw a conclusive correlation between amygdala volume and the onset of PTSD (Pieper et al., 2020; Ousdal et al., 2020). Inconsistent results of these studies could come from varying demographics in the studies (sex, race, type of trauma) and measuring the amygdala as a homogeneous rather than heterogeneous structure (Haris et al., 2023). The amygdala can be seen as both one whole nucleus as well as a structure consisting of multiple subnuclei, thus making it hard to determine whether it should be measured as one whole or multiple subparts. However, the many symptoms of PTSD corresponding with fear suggest it is critically involved in PTSD (Morey et al., 2012).

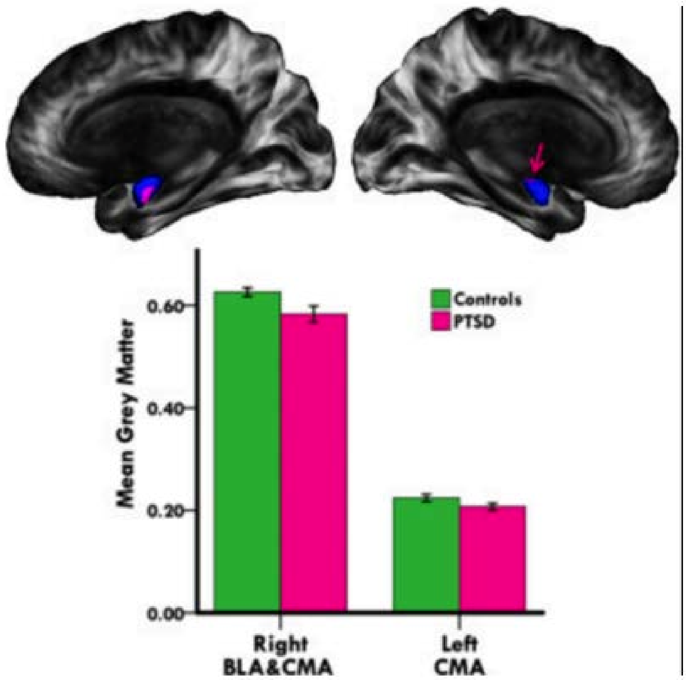


Figure 2. Abnormal amygdala volumes in adolescent PTSD patients. Bar graphs display smaller mean gray matter in both the basolateral amygdala and corticomedial amygdala for the right hemisphere in the PTSD patients. Left hemisphere displayed a smaller mean gray matter of the corticomedial amygdala in the PTSD patients.

Treatments for PTSD

Psychotherapy and pharmacology are both effective treatments for depressive disorders (Kamenov et al., 2017). There are also numerous studies done that have proven that healthy habits and practices such as exercise and diet greatly contribute to reducing PTSD symptoms as well (Schry et al., 2015; van den Berk-Clark et al., 2018; Correll et al., 2023). For example, Van der Kolk et al. published findings that yoga can greatly reduce PTSD symptomology and can help patients with PTSD control negative physical sensory experiences and overall functioning (2013). Treatments directly affecting the amygdala are limited, but one modern neurological technique called laser interstitial thermal therapy (LITT) or laser ablation has been shown to be effective (Patel & Kim, 2020). The surgical process of laser interstitial thermal therapy is outlined by Patel & Kim. The surgical procedure begins with an MRI or CT scan prior to surgery. Then, the optimal trajectory for the laser is planned using a computer-based navigation system. Once the patient is in the operating room, they are positioned properly to align with the laser probe, and a small stab incision is made at the planned entry site, followed by drilling a burr hole in the skull at the incision site. The laser probe is then inserted through the burr hole based on the pre-planned trajectory, and the patient is placed in an MRI scanner to verify probe position. When the probe is in position and secured, ablation can initiate, and periodic MRI images are taken. During the ablation, the laser emits photons, which are then absorbed by tumor chromophores (molecules in tumor cells that absorb light at specific wavelengths), releasing thermal energy. Once an ideal, elevated temperature is reached, proteins denature, cellular necrosis occurs, and tissue coagulates. After the ablation is completed and no other trajectories are planned, the patient is removed from the MRI scanner, the probe is removed, and the incision site is irrigated and closed. In a case study done in 2020 by Jon Willie et al., two patients with chronic PTSD underwent an amygdalohippocampectomy, a technique in which a neurosurgeon surgically removes the amygdala and/or hippocampus. The procedure targeted the amygdala in the right hemisphere and resulted in a reduced amount of seizures and a significant decrease in PTSD symptoms (Willie, 2020). The overall conclusion of this case series was that amygdalohippocampectomy can provide therapeutic healing for patients with PTSD, although it is a risky procedure.

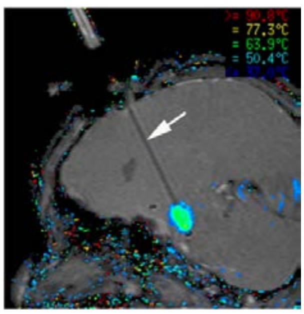


Figure 3. Laser interstitial thermal therapy uses a laser, shown by the white arrow, to ablate destructive or harmful regions of the brain such as tumors.

This method is still being developed and enhanced, such as innovations in laser probe design, probe cooling, and tissue temperature measuring technology (Patel & Kim, 2020). These developments continually make this procedure a more practical surgical technique and show promising indications to grow in the surgical field.

Conclusion

PTSD is a mental disorder that affects the amygdala, a fear-processing center and regulator for emotional responses. There are implications suggesting that PTSD affects amygdala physiology and volume, as it is often hyperactive in PTSD subjects. LITT is a promising treatment to treat patients with PTSD, and has potential to be more widely used as more research on its development continues.

References

1. Correll, T., Gentile, J., & Correll, A. (2023). Healthy Lifestyle Interventions Augmenting Psychotherapy in Anxiety and PTSD. Innovations in Clinical Neuroscience, 20(7-9), 18. https://pmc.ncbi.nlm.nih.gov/articles/PMC10561983/

2. Haris, E. M., Bryant, R. A., Williamson, T. H., & Korgaonkar, M. S. (2023). Functional connectivity of amygdala subnuclei in PTSD: a narrative review. Molecular Psychiatry. https://doi.org/10.1038/s41380-023-02291-w

3. Iribarren, J., Prolo, P., Neagos, N., & Chiappelli, F. (2005). Post-Traumatic Stress Disorder: Evidence-Based Research for the Third Millennium. Evidence-Based Complementary and Alternative Medicine, 2(4), 503–512. https://doi.org/10.1093/ecam/neh127

4. Johns, P. (2016, October 14). Functional neuroanatomy. Clinical Neuroscience. https://www.sciencedirect.com/science/article/pii/B9780443103216000035#s0140

5. Kamenov, K., Twomey, C., Cabello, M., Prina, A. M., & Ayuso-Mateos, J. L. (2017). The efficacy of psychotherapy, pharmacotherapy and their combination on functioning and quality of life in depression: a meta-analysis. Psychological Medicine, 47(3), 414–425. https://doi.org/10.1017/S0033291716002774

6. Morey, R. A., Gold, A. L., LaBar, K. S., Beall, S. K., Brown, V. M., Haswell, C. C., Nasser, J. D., Wagner, H. R., & McCarthy, G. (2012a, November). Amygdala volume changes in posttraumatic stress disorder in a large case-controlled Veterans Group Amygdala volume changes with post traumatic stress disorder in a large case-controlled veteran group. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3647246/

7. Ousdal, O. T., Milde, A. M., Hafstad, G. S., Hodneland, E., Dyb, G., Craven, A. R., Melinder, A., Endestad, T., & Hugdahl, K. (2020). The association of PTSD symptom severity with amygdala nuclei volumes in traumatized youths. Translational Psychiatry, 10(1). https://doi.org/10.1038/s41398-020-00974-4

8. Patel, B., & Kim, A. H. (2020). Laser interstitial thermal therapy. Missouri medicine. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7023945/

9. Pieper, J., Chang, D. G., Mahasin, S. Z., Swan, A. R., Quinto, A. A., Nichols, S. L., Diwakar, M., Huang, C., Swan, J., Lee, R. R., Baker, D. G., & Huang, M. (2020). Brain Amygdala Volume Increases in Veterans and Active-Duty Military Personnel With Combat-Related Posttraumatic Stress Disorder and Mild Traumatic Brain Injury. Journal of Head Trauma Rehabilitation, 35(1), E1–E9. https://doi.org/10.1097/htr.0000000000000492

10. Ressler, K. J. (2010). Amygdala activity, fear, and anxiety: Modulation by stress. Biological Psychiatry, 67(12), 1117–1119. https://doi.org/10.1016/j.biopsych.2010.04.027

11. van den Berk-Clark, C., Secrest, S., Walls, J., Hallberg, E., Lustman, P. J., Schneider, F. D., & Scherrer, J. F. (2018). Association between posttraumatic stress disorder and lack of exercise, poor diet, obesity, and co-occuring smoking: A systematic review and meta-analysis. Health Psychology, 37(5), 407–416. https://doi.org/10.1037/hea0000593

12. Schry, A. R., Rissling, M. B., Gentes, E. L., Beckham, J. C., Kudler, H. S., Straits-Tröster, K., & Calhoun, P. S. (2015). The Relationship Between Posttraumatic Stress Symptoms and Physical Health in a Survey of U.S. Veterans of the Iraq and Afghanistan Era. Psychosomatics, 56(6), 674–684. https://doi.org/10.1016/j.psym.2015.07.010

13. van der Kolk, B. A., Stone, L., West, J., Rhodes, A., Emerson, D., Suvak, M., & Spinazzola, J. (2013, November 14). Yoga as an Adjunctive Treatment for Posttraumatic Stress Disorder: A Randomized Control Trial. Trauma Research Foundation. https://traumaresearchfoundation.org/wp-content/uploads/2021/05/Yoga-F-J-Clin-Psychiat-1.pdf

14. Willie, C. J. T. (2020, October). Case series: Unilateral amygdala ablation ameliorates...: Neurosurgery. CNS Neurosurgery. https://journals.lww.com/neurosurgery/fulltext/2020/10000/case_series__unilateral_amygdala_ablation.23.aspx

About the Author

Micah is a freshman at UIUC majoring in neuroscience. He became involved in Brain Matters to gain experience writing research articles. Outside of academics, you can find him staying active in the ARC by working out or playing volleyball. In the future, Micah hopes to make it to medical school one day.

The Cognitive Neuroscience of Moral Decision-Making in Extreme Situations: High-Stress or Life-Threatening Scenarios

Written by May Yang

Abstract

Scientists have researched the intricacies of cognitive neuroscience in an attempt to explain how extreme stress changes moral decision-making in critical life-or-death situations. By examining the complicated interactions among key brain regions–the amygdala, ventromedial prefrontal cortex (VMPFC), dorsolateral prefrontal cortex (DLPFC), and anterior cingulate cortex (ACC)–we uncover how increases in cortisol and adrenaline modulate the shift from ethical reasoning to base instinctual reactions. Compelling research and neuroimaging results have revealed how high-stress situations amplify emotional reactions while impairing rational thinking, leading to impulsive decisions that favor self-preservation over morality. Such findings are in direct opposition to leading views regarding moral reasoning and make a strong case for the urgent need for strategies to improve ethical decision-making in high-pressure situations–especially among individuals standing at the forefront when crises occur.

I. Introduction

Imagine a moment when your entire being jolts to attention–your blood surges through your veins, every cell in your body tingles. Suddenly, you're moving, acting, driven by some primal force beyond your conscious control. Afterward, you're dazed, trying to recall the hazy sequence. You question your actions. This is the instinct in control–decision-making under extreme situations. Decision making, defined by the American Psychological Association (APA), refers to the cognitive process of choosing between two or more alternatives (APA, 2018). But when we assume that every decision made could mean life or death, the situation becomes less simple. In the blink of an eye, a firefighter must choose: rush into a collapsing building to save lives, or stay back and live another day. This split-second moral dilemma under extreme pressure shatters the comfortable paradigms of Kahneman's dual-process theory. While we leisurely ponder between a sandwich or burger, tapping into our System 1 or System 2 thinking, these heroes face a crucible where morale and morality collide (APA, 2018). This raises the central question of this article: How does extreme stress influence the brain's moral decision-making?

II. Neurobiological Foundations

The brain is like a puzzle. Each piece contains its own unique details and meaning, but it's only when these pieces are put together that they provide a full picture. The key components involved in moral decision-making are: the amygdala, ventromedial prefrontal cortex (VMPFC), dorsolateral prefrontal cortex (DLPFC), temporoparietal junction (TPJ), anterior cingulate cortex (ACC), and posterior cingulate cortex (PCC). Beginning with the amygdala–a small almond-shaped structure located in the medial temporal lobe (Salzman, 2024). The amygdala is the fastest brain component to react to stress when anything may potentially threaten one's life. It is connected to the thalamus, so input from sensory systems will relay to the amygdala which can trigger a response ready for danger, signaling the body to initiate a fight-or-flight response. It works as a counterforce to rational thinking and decision-making, allowing individuals to prioritize their own survival (Mendez, 2009). If we primarily relied on the amygdala's reaction, it would mean we could never make moral decisions in life-threatening situations, but that is not the case. This is where the VMPFC comes into play. The VMPFC helps balance emotions and rationality. It anticipates potential outcomes, then decides whether to prioritize personal safety or saving others (Yoder and Decety, 2018; Mendez, 2009).

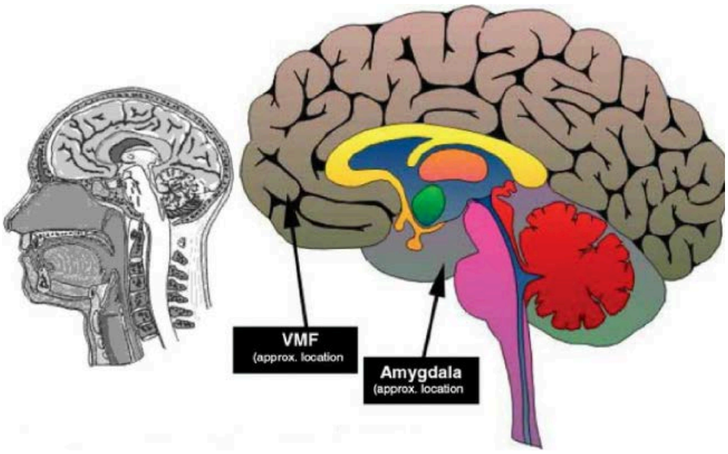


Figure 1: The Amygdala and vmPFC Brain Regions (Walls et al., 2011)

The dorsolateral prefrontal cortex (DLPFC) also plays a crucial role by helping evaluate the long-term consequences of decisions, ensuring that the individual isn't driven purely by short-term emotional reactions (Baumgartner et al., 2013). Meanwhile, the anterior cingulate cortex (ACC) mediates conflicts between emotion and logic, helping to reconcile the often-competing interests of the amygdala and the prefrontal cortex (Mendez, 2009). Together, these regions create a dynamic network where rationality, emotion, and survival instincts are constantly negotiating the best course of action, particularly under stress. This seamless interaction explains how humans can make complex moral decisions even when faced with imminent danger.

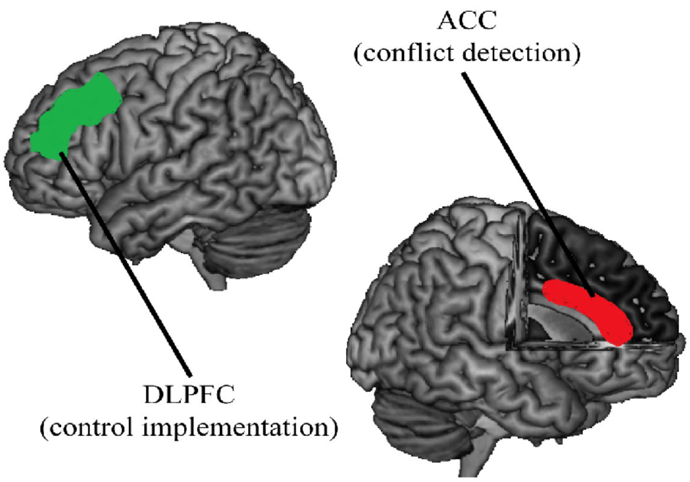


Figure 2: ACC and DLPFC Brain Regions (Torres-Quesada, 2013)

III. Impact of Extreme Stress on the Brain

Stress institutes a cascade of physiological reactions that cause the brain to function differently during moral decision-making. During extreme levels of stress, the brain releases large amounts of cortisol and adrenaline, two stress hormones designed to prepare the body for a rapid response. These surges of hormones, however, strongly influence the different brain regions' operation.

The prefrontal cortex, which is responsible for rational thinking and long-term decision-making, is significantly compromised in the event of high levels of stress. For instance, cortisol has been found to diminish the functioning of the PFC, which makes it rather difficult to deliberate a complex decision or to weigh consideration against long-term consequences (Arnsten, 2009). At the same time, there is an increase in the activity level of the amygdala, hence giving rise to increased emotional response as well as giving way to immediate reactions based on survival instincts rather than ethical ones (Ochsner et al., 2009). The resulting imbalance between the PFC and amygdala tips the brain toward emotionally-laden instinctive action, bypassing careful deliberation that is necessary for moral judgments.

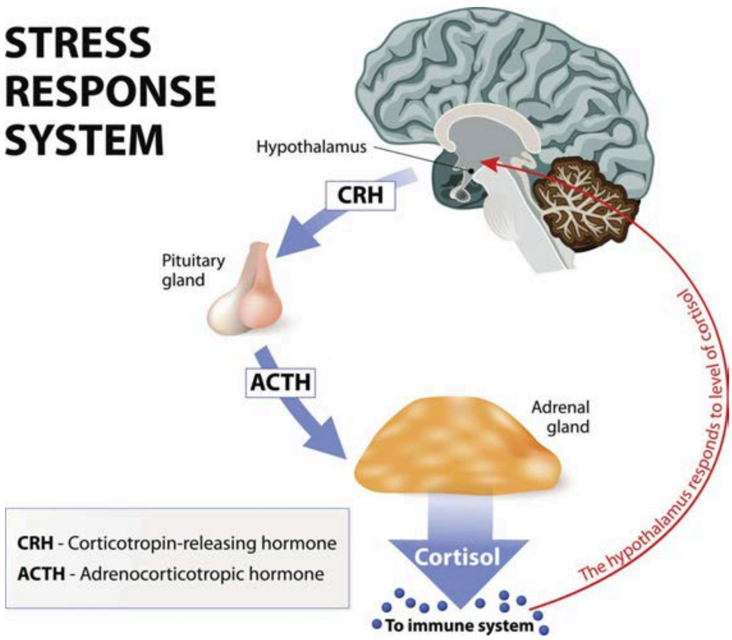


Figure 3: Cortisol's Flow and Impact (Russell & Lightman, 2019)

Further, the ACC overworks itself trying to moderate the conflict between these parts of the brain. Stressed-induced disturbances in the brain make it more arduous for the ACC to promote clear decision-making, thus pushing the person toward faster and often emotionally rationalized choices. In extreme stress, moral decision-making is heavily biased toward emotional reactions rather than reason-based judgment, which underlines the fragility of our ethical reasoning under pressure.

IV. Empirical Evidence: Studies on Stress and Moral Choices

Several laboratory experiments have revealed decisive evidence of how moral decision-making is distorted by stress. In these controlled experiments, participants are exposed to simulated conditions of stress such as public speaking or physical discomfort, in which circumstances subjects are more likely to favor immediate and emotionally driven responses when faced with dilemmas. For example, studies that put their participants in high levels of stress showed that participants are more likely to depend on intuition when solving complex moral dilemmas rather than ethical reasoning for human survival or their safety (Starcke et al., 2008).

Neuroimaging allows a glimpse of the activity of the brain involved in such decisions. fMRI scans revealed that during stressful conditions, decisions resulted in decreased activity of the prefrontal cortex along with increased amygdala activation. Such findings support the hypothesis that the cognitive load created by stress impairs the brain's ability to sustain or otherwise continue its state of rational ethical thinking. Instead, the hypertrophied activity of the amygdala enhances emotional responses and motivates individuals toward behaviors that fulfill the immediate need for survival or emotional gratification (Shin et al., 2005).

One of the clear findings that come out of these studies is that under intense stress, moral calculus, as it normally runs in the brain, tends to get disrupted. People are more likely to make decisions that have to do with self-preservation rather than broader ethical concerns – a powerful illustration of how deeply stress embeds its influence on shaping moral behavior.

“Under intense stress, moral calculus, as it normally runs in the brain, tends to get disrupted.”

V. Real-World Applications

The implications of cognitive neuroscience’s impact on moral-decision making are great for professions that face high levels of stress: healthcare workers, soldiers, firefighters, and law enforcement officers. These professionals make sound ethical decisions during life-and-death situations. Training programs inculcating stress management techniques would help professionals maintain the cognitive clarity to navigate these moral dilemmas effectively. Organizations could also implement ethical policies or procedures that consider how much stress modifies decision-making, such as reviewing decisions made under extreme tension orembedding procedures that may mitigate the effects of stress on moral judgment.

Furthermore, this knowledge can enhance technology, particularly AI systems. By embedding models that describe how human decision-making changes under stress, AI algorithms–such as those used in autonomous driving systems–could be configured in a way that takes into consideration subtle, stress-driven behavior of human operators.

VI. Conclusion

Extreme stress causes a critical disruption of the balance between emotion and reason within the brain during moral decision-making. It is this delicate balance in the interplay between the amygdala, prefrontal cortex, and anterior cingulate cortex that becomes imbalanced, resulting in decisions driven by survival and emotion rather than reasoned ethical judgment. Awareness of these neural mechanisms in high-stress settings may inform strategies for supporting better moral decision-making by individuals and organizations. This implication requires further research about stress and the brain, so that the systems and structures we rely on during crises are equipped to make morally sound decisions when overwhelming stress occurs.

References

1. American Psychological Association. (2018). Moral decision making. APA Dictionary of Psychology. https://dictionary.apa.org/moral-decision-making

2. APA. (2024). APA Dictionary of Psychology. Apa.org. https://dictionary.apa.org/decision-making

3. APA. (2024). APA Dictionary of Psychology. Apa.org. https://dictionary.apa.org/dual-process-theory

4. Arnsten, A. F. (2009). Stress signaling pathways that impair prefrontal cortex structure and function. Nature Reviews Neuroscience,10(6), 410-422. https://doi.org/10.1038/nrn2648

5. Baumgartner, T., Knoch, D., Hotz, P., Eisenegger, C., & Fehr, E. (2013). Dorsolateral and ventromedial prefrontal cortex orchestrate normative choice. Nature Neuroscience, 16(9), 1251-1253. https://doi.org/10.1038/nn.3445

6. Greene, J. D., Nystrom, L. E., Engell, A. D., Darley, J. M., & Cohen, J. D. (2004). The neural bases of cognitive conflict and control in moral judgment. Neuron, 44(2), 389-400. https://doi.org/10.1016/j.neuron.2004.09.027

7. McEwen, B. S. (2012). Brain on stress: How the social environment gets under the skin. Proceedings of the National Academy of Sciences, 109(Supplement_2), 17180-17185. https://doi.org/10.1073/pnas.1121254109

8. Mendez, M. F. (2009). The neurobiology of moral behavior: Review and neuropsychiatric implications. CNS Spectrums, 14(11), 608-620. https://doi.org/10.1017/S1092852900023769.
Ochsner, K. N., & Gross, J. J. (2009). The neural architecture of emotion regulation. Handbook of Emotion Regulation, 38, 87-106.
https://doi.org/10.1093/acprof:oso/9780195311353.003.0004
10. Russell, G., & Lightman, S. (2019). The human stress response. Nature Reviews Endocrinology, 15(9), 525–534. https://doi.org/10.1038/s41574-019-0228-0
11. Salzman, C. Daniel (2024). amygdala. Encyclopedia Britannica.
https://www.britannica.com/science/amygdala
12. Shin, L. M., Wright, C. I., Cannistraro, P. A., Wedig, M. M., McMullin, K., Martis, B., Macklin, M. L., Lasko, N. B., Cavanagh, S. R., Krangel, T. S., Orr, S. P., Pitman, R. K., Whalen, P. J., & Rauch, S. L. (2005). A functional magnetic resonance imaging study of amygdala and medial prefrontal cortex responses to overtly presented fearful faces in posttraumatic stress disorder. Archives of General Psychiatry,62(3), 273–281. https://doi.org/10.1001/archpsyc.62.3.273
13. Starcke, K., & Brand, M. (2012). Decision making under stress: A selective review. Neuroscience & Biobehavioral Reviews, 36(4), 1228-1248. https://doi.org/10.1016/j.neubiorev.2012.02.003
14. Torres-Quesada, Maryem. (2013). Reactive and proactive modulation in cognitive and emotional control.
Walls, Andrew, Okumus, Fevzi, & Wang, Youcheng. (2011). Cognition and Affect Interplay: A Framework for the Tourist Vacation Decision-Making Process. Journal of Travel & Tourism Marketing, 28, 567-582. https://doi.org/10.1080/10548408.2011.588121
15. Yoder, K. J., & Decety, J. (2018). The neuroscience of morality and social decision-making. Annual Review of Psychology, 69, 101-127. https://doi.org/10.1146/annurev-psych-122216-011403

May Yang
May is a freshman at the University of Illinois majoring in Brain & Cognitive Science. She joined Brain Matters to learn more about the brain, develop her research skills, and connect with people who share similar interests. In addition to writing for Brain Matters, May is also involved in Dr. Hotaling's Cognitive Decision Making Lab. In the future, she hopes to pursue a PhD in a related field.

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President

Michelle Bishka

Michelle Bishka is a senior majoring in Specialized Chemistry and minoring in Computer Science. Outside of Brain Matters, she is an undergraduate researcher in the Silverman Lab and a member of American Chemical Society. She later hopes to pursue graduate studies in chemistry.

Chief Editor

Andrew Hamilton

Andrew Hamilton is a junior with a major in Neuroscience and minors in Spanish and Chemistry. One thing he enjoys about editing is that he gets to read so many interesting articles about science-related discoveries every day! Outside of the club, he pursues research regarding optimization with on-tissue chemical derivatization.

Vice President

Praise Kim

Praise Kim is the Vice President of Brain Matters and an undergraduate researcher pursuing a BSLAS in Brain and Cognitive Science. Currently, as a research assistant in the Gratton Lab, she studies the Fronto-Parietal Network in cognitive control tasks across different mental states. In the past, she has also presented work on the infant parasympathetic response and maternal depression with the Interdisciplinary Lab for Social Development. She is broadly interested in cognition in the brain and throughout development, also presenting work on social cognitive development at Stanford University. Outside of research, she lifts weights, reads fantasy novels, and spends time with her church. Her future goals are to continue researching the brain—whether as a post-bacc, doctoral student, post-doc, or professor.

Assistant Chief Editor

Krisha Agarwal

Krisha Agarwal is a junior in MCB Honors with a minor in Informatics. She is the Editor-in-chief of Brain Matters and a member of American Medical Women's Association. She is also an undergraduate researcher at the KV Prasanth Lab in Cell and Developmental Biology. In the future, she hopes to attend graduate school and work in the biotechnology industry. In her free time, Krisha enjoys crocheting, reading, sketching, and spending time with friends.

Assistant Chief Editor

Macy Hoeveler

Macy Hoeveler is a sophomore in the Brain & Cognitive Science program at UIUC. She is pursuing a double minor in Integrative Biology and Music. Aside from being the Editor-in-Chief of Brain Matters, she is a writing consultant with the Writer's Workshop. In addition, she is a Beckman Fellow with the Auditory Cognitive Neuroscience Lab and a lab assistant at the Dolezal Bee Research Lab. In her free time, Macy is a violinist in the Philharmonia Orchestra and enjoys reading, listening to music, and collecting bugs. She hopes to continue pursuing biology in graduate school, studying behavioral genetics and neurobiology.

Treasurer

Vraj Patel

Vraj Patel is a sophomore majoring in Neuroscience with minors in Chemistry and Psychology. Vraj joined Brain Matters to learn about more niche topics in neuroscience and research in the field. In addition to being treasurer for Brain Matters, Vraj is an undergraduate researcher in the Sweeney Lab, which studies neuroscience in the context of feeding and related behaviors. He is also a volunteer for Avicenna Community Health Center, a course assistant for STAT 200, and a peer mentor for first-year students in the neuroscience major. Vraj hopes to explore more in the field of neuroscience from a medical perspective in the future!

Social Media Chair

Vani Sharma

Vani Sharma is pursuing a Bachelor of Science in Molecular and Cellular Biology (MCB) with an honors concentration, alongside a minor in Public Health and a Neuroscience certificate. As a writer for Brain Matters, she investigates the intricate interplay between the brain and diverse phenomena, including the neural foundations of gratitude, the influence of music on cognitive processes, and the complexities of neuroanatomy and neurological disorders. Through her work, she blends rigorous scientific research with engaging narratives to illuminate the brain's extraordinary intricacies while promoting scientific literacy and making complex concepts accessible to a broader audience.

Social Media Chair

Erin Ford

Erin Ford is a junior majoring in Chemical Engineering with a concentration in Biomolecular Engineering. In her free time, she enjoys playing tennis and painting. She hopes to help others increase their knowledge about neuroscience through her writing in Brain Matters.

Social Chair

Isabelle Afshari

Isabelle is a sophomore at the University of Illinois majoring in Neuroscience. Isabelle became involved in Brain Matters to learn more about writing scientific articles and innovations in neuroscience. In addition to writing for Brain Matters, Isabelle is involved in McKinley Health Stress Management Peers, LAS Leaders, and Women's Glee Club. In the future, Isabelle hopes to attend medical school and continue reading and writing about new scientific innovations!

Social Chair

Kaitlyn Tuvilleja

Kaitlyn Tuvilleja is a junior in Bioengineering with a Statistics minor. She is an undergraduate research assistant for Bhargava Lab and I^2 Lab. Besides Brain Matters, Kaitlyn is involved with SWE, WIE, and BMES. In her spare time, she enjoys baking and running with her friends.

Design Head

Sarah Masud

Sarah is a junior studying Psychology and Information Sciences with a minor in Art & Design. Some of her academic interests include cognition, human-computer interaction, and treating psychiatric disorders. She enjoys drawing, finding new music, and crocheting as well! Outside of Brain Matters, Sarah is also involved in Design Innovation Illinois and Psi Eta Mu, a professional information sciences fraternity. She hopes to continue furthering her understanding of neuroscience and exploring topics she's passionate about through the journal.

Design Board

Ruth Anderson

Ruth Anderson is a rising Sophomore at the University of Illinois majoring in neuroscience and minoring in psychology. She joined Brain Matters to get involved with the neuroscience community on campus and learn more about the field. Ruth currently is hoping to pursue a career in research. She is passionate about womens health and child development. Outside of school Ruth enjoys hanging out with her friends, crocheting, and reading.

Esther Nam

Esther Nam is a junior on the pre-medical track majoring in Psychology with a minor in Public Health. She is interested in exploring the cognitive and neurological impacts of bilingualism, and is currently a research assistant in the Educational Psychology Psycholinguistics Lab with a focus on cognitive psych. In her free time, she loves to draw, play games, and spend time with friends. After undergrad, Esther hopes to attend medical school to become a physician.

Jeslyn Chen

I'm a rising sophomore majoring in psychology and minoring in chemistry. I joined Brain Matters to combine my interests of neuroscience, psychology, and journalism. Outside of this magazine, I plan to become a student EMT at UIUC and enjoy drawing, going to concerts, and thrifting.

Jessica George

Hi! My name is Jessica George and I'm a junior majoring in Molecular and Cellular Biology and Brain and Cognitive Science. Outside of school, I volunteer at a nursing home in the activities department, where I work closely with residents who have dementia. In my free time I love dancing, listening to music, and trying new restaurants!

Lisa Patel

Lisa Patel is a rising junior and an Integrative Biology major on the pre-medical track with minors in Chemistry and Nutrition at UIUC. Passionate about medicine and community outreach, she co-founded and serves as President of the Illini Sheltering Hands Society, where she teaches basic life-support skills and organizes volunteering initiatives. As Public Relations Coordinator for REACT, Lisa coordinates hands-on chemistry demonstrations at local elementary and middle schools. She's volunteered over 300 hours at UI Health Hospital while assisting across Emergency, Diagnostics, Radiology, University Health Services, and Surgical departments. She also directs community health initiatives as Director of Medicine for UIUC's MEDLIFE chapter. Her end goal is to become a physician and she is dedicated to expanding her knowledge to better serve her community.

Sania Shah

Sania Shah is a sophomore majoring in Brain and Cognitive Science with a minor in Data Science. She works as an undergraduate research assistant in the Cognitive Decision-Making Lab and dances competitively with the Illini Raas team. Outside of Brain Matters, Sania enjoys playing badminton with friends and curling up with a good book and an iced coffee.

Editors

Thiya Ilankovan

Thiya is a sophomore at UIUC majoring in MCB with a minor in Psychology, hoping to one day become a Physician Assistant. In her free time, she likes to run, crochet, and play the piano. She is currently involved in research at the Liang Lab for Behavioral Neuroscience. Through her involvement with Brain Matters, she hopes to broaden her knowledge and gain deeper insights into the fields of neuroscience and psychology.

Kathryn Kennedy

Kathryn Kennedy is a freshman studying Biology with minors in Health Technology and Spanish. She joined Brain Matters to learn more about neuroscience, psychology, and improve her writing and editing skills. Outside of the journal, she is involved in Global Medical Training and Education and Training 4 Health. She also dances with PSA Barkada, sings with the St. John's church choir, and plays guitar in her free time. Her career goal is to be a pediatrician.

Nicholas Opiola

Nicholas Opiola is a recent ’24 MCB alumni. He is a lifelong learner and has always loved studying across all academic disciplines, especially neuroscience! Nicholas joined Brain Matters to immerse himself in all the latest exciting work being performed in the field of neuroscience and to utilize his writing skills towards helping others produce their best work. In his free time, Nicholas loves to watch fútbol, dance, sing karaoke, spend time with family and close friends, play video games, and spend time amongst nature. In the future, Nicholas hopes to devote his career towards making a lasting, positive change in as many lives as possible.

Megan Lu

Megan Lu is a Junior majoring in Brain & Cognitive Science with a minor in Health Administration and Business. She is involved in various RSOs on campus, including FHCE (Future Healthcare Executives) and Alpha Epsilon Delta (a pre-health fraternity). She is also currently involved in research with the Illinois Alternative Protein Project. In her free time, Megan spends most of her time at the gym working out, cooking new recipes, or listening to true crime podcasts. She hopes to deepen her understanding and appreciation of the brain through writing with Brain Matters and will graduate this year.

Yuliia Kohut

Yuliia Kohut is a Freshman in Bioengineering on a pre-medical track and a student from Ukraine. Apart from Brain Matters, on campus she is a Global Health executive member in the American Medical Student Association, and she is also a student volunteer at Carle Hospital. Yuliia is an undergraduate researcher in Dr. Best-Popescu lab at Beckman Institute, working on developing imaging tools for cellular neuroscience research. In her free time Yuliia enjoys cross-stitching, cooking Ukrainian food, and reading sci-fi novels. She joined the editing and writing team of Brain Matters to share her fascination with neuroscience with UIUC!

Kaitlyn Tuvilleja

Kaitlyn Tuvilleja is a junior in Bioengineering with a Statistics minor. She is an undergraduate research assistant for Bhargava Lab and I^2 Lab. Besides Brain Matters, Kaitlyn is involved with SWE, WIE, and BMES. In her spare time, she enjoys baking and running with her friends.

Gus Dorman

Gus Dorman is a freshman majoring in Neuroscience with a minor in Computer Science. He joined Brain Matters as an editor to learn more about the field while also getting a feel for what research articles are like. If he's not studying, he's probably longboarding around campus, playing a video game, or watching shows.

Praise Kim

Praise Kim is the Vice President of Brain Matters and an undergraduate researcher pursuing a BSLAS in Brain and Cognitive Science. Currently, as a research assistant in the Gratton Lab, she studies the Fronto-Parietal Network in cognitive control tasks across different mental states. In the past, she has also presented work on the infant parasympathetic response and maternal depression with the Interdisciplinary Lab for Social Development. She is broadly interested in cognition in the brain and throughout development, also presenting work on social cognitive development at Stanford University. Outside of research, she lifts weights, reads fantasy novels, and spends time with her church. Her future goals are to continue researching the brain—whether as a post-bacc, doctoral student, post-doc, or professor.

Jessica Chen

Jessica Chen is a sophomore studying clinical-community psychology. With Brain Matters, she has been excited to integrate her interests in neuroscience, linguistics, and psychology. She has appreciated groundbreaking applications of neuroscience in skill acquisition, discrimination, addiction, and more. Currently a research assistant with the Health Equity and Action Lab and the Social Cognition Lab, Jessica examines parenting and child health outcomes across cultural contexts, and neural network dissection of trends in biases. Aside from academics, Jessica is most likely baking a sweet treat or lounging at a matcha cafe.

Natalia Pacheco

Natalia is a Freshman at the University of Illinois majoring in Neuroscience. Natalia became involved in Brain Matters to further her passion for the brain and to become familiar with modern topics of neuroscience. In addition to Brain Matters, Natalia is involved in the American Medical Women's Association at the University of Illinois. Natalia hopes to continue her studies in the medical field specifically with neurology to continue learning about the brain!

Brain Matters Writers

Noreen Adoni

Noreen is a freshman at the University of Illinois majoring in Neuroscience. She joined Brain Matters to investigate how the brain impacts the ways in which we interact with the world around us and stay updated on current research in the field. Noreen is interested in studying neurological diseases, hoping to further analyze and treat them as a physician in the future.

Alexander Byrne

Alexander Byrne, a native of the South Side of Chicago, has long fostered his fascination with science and narrative. Now a student pursuing a degree in Neuroscience, Alexander has focused his scholarly work on the overlap of maternal immune activation, neurodevelopmental disorders, and gut-brain microbiota interactions with the aim of determining how early immune signals influence the development of the brain and long-term behavioral outcomes.In addition to this study, he is deeply engaged in molecular neuroscience investigations into the structural processes of prion protein misfolding and aggregation. After finishing his undergraduate studies, Alexander plans to undertake a Ph.D. in neuroscience. His desire is to be involved in translational research that converts molecular biology into clinical knowledge.

Anika Chandola

Anika Chandola is a Freshman Majoring in MCB with a minor in Psychology and Chemistry on the Pre-Med track. She is currently working in Bagchi Lab researching the environmental impact on reproductive health and volunteered at UChicago's Phlebotomy Clinic. She is also a member of the Gamma Phi Beta Sorority working alongside Girls on the Run. In her freetime she enjoys fashion, swimming, pageants and playing the piano. She hopes to become more involved in the neuroscience field and learn more about this diverse community.

Rayyan Iqbal

Rayyan Iqbal is a sophomore at the University of Illinois, majoring in Chemistry. He is currently conducting research in the Physical Activity and Neurocognitive Health Lab, where he studies the impact of physical behaviors—such as physical activity and sedentary time—on brain health. Beyond his research, Rayyan is actively involved in REACT, an outreach program that brings science to life for young students in the Champaign-Urbana area.

Isha Kandlikar

Isha Kandlikar is a rising junior at the UIUC majoring in Molecular and Cellular Biology with minors in Business and Public Health. She is an undergraduate researcher in the Rudolph Lab, where she studies genetic mouse models to explore treatments for psychiatric disorders. Outside the lab, Isha is a marketing and healthcare consultant, a member of the Illini Medical Screening Society, and involved in Alpha Epsilon Delta, a pre-health fraternity on campus. Passionate about neuroscience, she is excited to write for Brain Matters and dive deeper into specific topics within the field.

Yuliia Kohut

Yuliia Kohut is a Freshman in Bioengineering on a pre-medical track and a student from Ukraine. Apart from Brain Matters, on campus she is a Global Health executive member in the American Medical Student Association, and she is also a student volunteer at Carle Hospital. Yuliia is an undergraduate researcher in Dr. Best-Popescu lab at Beckman Institute, working on developing imaging tools for cellular neuroscience research. In her free time Yuliia enjoys cross-stitching, cooking Ukrainian food, and reading sci-fi novels. She joined the editing and writing team of Brain Matters to share her fascination with neuroscience with UIUC!

Brianna Mae Huner

Brianna Mae is a Junior at the University of Illinois majoring in Clinical/Community Psychology. She became involved in Brain Matters to gain more experience researching and writing about the current research in Neuroscience. When she is not writing for Brain Matters, she is also involved in Dr. Kwapil's Project on Life Experiences Lab, and is the Treasurer for the Psychology Research and Community Club (PRACC). Brianna Mae is hoping to pursue a PhD in Clinical Neuropsychology and conduct research about the neurological basis behind different clinical disorders.

Emily Aldrich

Emily Aldrich is a Freshman majoring in Neuroscience with minors in Linguistics and Psychology on the pre-med track. Emily joined Brain Matters to gain a deeper understanding of the brain through exploring current research topics in neuroscience. In her free time, she enjoys listening to music, reading, and spending time with friends.

Leah Rupp

Leah Rupp is a freshman at the University of Illinois in Urbana-Champaign studying Molecular and Cellular Biology within the honors concentration. Leah joined Brain Matters to get the opportunity to learn and write about new neuroscience research. Leah is also a Stress Management Peer with McKinley Health Center and a volunteer with the Food Assistance and Well-being Program. In her free time, Leah enjoys running and playing the piano. Her career aspiration is to become a physician.

Ananya Sampathkumar

Ananya Sampathkumar is a sophomore, majoring in Neuroscience with minors in Chemistry and Public Health. Outside of Brain Matters, Ananya is an assistant editor-in-chief for Double Helix Digest, a member of Starcourse, a volunteer at Carle Hospital, and works at the Office of Undergraduate Admissions as a tour guide and student ambassador. In her free time, Ananya likes to read books, make jewelry, watch movies, and hang out with her friends.

Vani Sharma

Vani Sharma is pursuing a Bachelor of Science in Molecular and Cellular Biology (MCB) with an honors concentration, alongside a minor in Public Health and a Neuroscience certificate. As a writer for Brain Matters, she investigates the intricate interplay between the brain and diverse phenomena, including the neural foundations of gratitude, the influence of music on cognitive processes, and the complexities of neuroanatomy and neurological disorders. Through her work, she blends rigorous scientific research with engaging narratives to illuminate the brain's extraordinary intricacies while promoting scientific literacy and making complex concepts accessible to a broader audience.

Navi Singh

Navi Singh is a rising Junior at the University of Illinois Urbana-Champaign, majoring in Neuroscience and minoring in Health Administration and Chemistry on the Pre-Medical track. She is an aspiring physician hoping to specialize in Neurology. She is also involved with several RSO's on campus. These include serving as Social Chair for Udaan and the Undergraduate Neuroscience Society, being a member of a Pre-Health Professional Fraternity Phi Chi, mentor for Illini Mentor Program, volunteer for Global Medical Brigades, and Research Assistant at the Kukekova lab. Navi is excited to share her first article and research with Brain Matters!

Pravika Srivastava

Pravika Srivastava is a rising junior at the University of Illinois Urbana-Champaign majoring in neuroscience with a minor in psychology on the pre-medical track. She is passionate about brain health, mental well-being, and hopes to pursue a career in psychiatry. As a writer and new Social Media Co-Chair for Brain Matters, she enjoys writing about neuroscience-related topics while eager in helping expand the journal's outreach. On campus, Pravika volunteers in the Pediatric ICU at Carle Foundation Hospital, conducts research at the CONNECTlab and Rudolph Lab, and serves on the Speaker Committee for Alpha Epsilon Delta. Pravika is excited to share her research and writing as part of her ongoing commitment to advancing understanding of the brain and mental health.

Micah Wang

Micah is a freshman at UIUC majoring in neuroscience. He became involved in Brain Matters to gain experience writing research articles. Outside of academics, you can find him staying active in the ARC by working out or playing volleyball. In the future, Micah hopes to make it to medical school one day.

May Yang

May is a freshman at the University of Illinois majoring in Brain & Cognitive Science. She joined Brain Matters to learn more about the brain, develop her research skills, and connect with people who share similar interests. In addition to writing for Brain Matters, May is also involved in Dr. Hotaling's Cognitive Decision Making Lab. In the future, she hopes to pursue a PhD in a related field.

Want To Get Involved?

Brain Matters is a Registered Student Organization (RSO) on campus that welcomes all years and majors. Please email brainmattersuiuc@gmail.com with inquiries about getting involved with the journal or RSO.